ARCHIVES OF OPHTHALMOLOGY

VOLUME 19 (old series Volume 76) JUNE 1938 NUMBER 6

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PERSONAL EXPERIENCES WITH INTRACAPSULAR CATARACT EXTRACTIONS

FREDERICK ALLISON DAVIS, M.D.

MADISON, WIS.

The intracapsular method of cataract extraction is increasing in popularity both in this country and abroad, though it has by no means supplanted the extracapsular procedure. While some ophthalmologists still reject the operation because of convictions concerning the wisdom of removing the posterior capsular diaphragm and the anatomic disturbance of the vitreous body, many have hesitated because of fear of the operation. From my limited experience, the latter seems entirely unjustified. The impression has prevailed that the intracapsular method is too hazardous for any but surgeons of large experience, and many men have avoided the operation for this reason. No doubt prejudice against intracapsular extraction has been due to early unfortunate results reported in this country following the use of the original method introduced by Colonel Smith. With the more refined and gentle technic of forceps extraction, this should be dispelled.

In this paper are reported the results of 200 consecutive intracapsular cataract extractions performed by me and by the members of the staff of the Wisconsin General Hospital during a period of four years. In about two thirds of the cases the operation was performed by me personally, and in the remainder, by associates, assistants and resident staff members. Thus, in considerable number the first intracapsular operations performed by members of the resident staff are included. This point is stressed in order that the reader may properly evaluate the results.

The operations herein reported were performed by the combined forceps and expression technic, similar to the methods originally intro-

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From the Department of Ophthalmology, University of Wisconsin Medical School.

Read at a meeting of the American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 13, 1937.

Readers interested in the discussions on this article are referred to the Transactions of the American Academy of Ophthalmology and Otolaryngology (**42**: 235, 1937).

duced by Stanculeanu, Knapp and Török and modified by Lancaster, Šafář, Elschnig and others. There is, however, a definite variation or modification of their procedures, to be described later. It is beyond the scope of this paper to enter on a detailed description of the evolution of the forceps expression method of intracapsular extraction. This has been adequately covered by Lancaster.[1]

There was no rigid rule for the selection of patients for operation, though those with complications, such as a high degree of myopia, an increase in intraocular tension, fluid vitreous or posterior synechiae, were usually, though not invariably, subjected to extracapsular extraction.

PREPARATION FOR OPERATION

Complete physical examination and Wassermann tests were given practically all patients. All foci of chronic sepsis were removed when possible. This pertained especially to abscessed teeth, pyorrhea and similar conditions. In those cases in which the lacrimal sac showed secretion or in which there was a history of recurrent infection, extirpation was performed prior to operation.

Smears from the conjunctiva of the lids were invariably made, and operation was rarely performed unless they were negative for bacteria and pus cells.

About 18 per cent of the patients were operated on under avertin anesthesia, the technic of which has been discussed by me elsewhere.[2] Those patients operated on under local anesthesia received 1½ grains (0.09 Gm.) of pentobarbital sodium by mouth one-half hour prior to operation. One drop of a 3 per cent solution of euphthalmine hydrochloride was instilled into the conjunctival sac one hour before operation. The eyebrow was shaved or clipped.

TECHNIC

Operation.—Preparation on the operating table consisted of the usual thorough cleansing of the skin about both eyes and the painting of the skin of the lids, brow and nose with a 3 per cent tincture of iodine. This was usually wiped off with 70 per cent alcohol. The conjunctival sac was thoroughly irrigated with boric acid solution, and the lids were cleansed with a solution of mercury bichloride in a concentration of 1 : 5,000. From 5 to 6 drops of a 4 per cent solution of cocaine hydrochloride was instilled into the cul-de-sac over a period of twenty-five minutes. One or 2 drops of epinephrine hydrochloride in a concentration of 1 : 1,000 was instilled just before operation. Akinesia was produced according to the method of Van Lint. A 2 per cent solution of procaine hydrochloride was generally used, an injection also being made into the orbicularis muscle at the internal canthus. One drop of a 1 per cent solution of cocaine was injected subconjunctivally at the limbus below. Retro-orbital injection of procaine hydrochloride through the skin along the floor of the orbit was used in about 25 cases, but this was reserved for use in special instances.

A speculum was invariably used throughout the operation, Skeel's modification of Weeks' speculum being preferred in most cases.

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1. Lancaster, W. B.: The Cataract Operation, Surg., Gynec. & Obst. **52**:452 (Feb.) 1931.

2. Davis, F. A.: Tribromethanol (Tribromethyl-Alcohol, Avertin) as an Anesthetic in Eye Surgery, Tr. Am. Ophth. Soc. **29**:47, 1931.

Fixation of the globe was made at the lower margin of the internal rectus muscle at its insertion, an ordinary fixation forceps with a catch being used. A bridle suture under the superior rectus muscle was used in about half of the cases. A Graefe incision of from 35 to 45 per cent of the corneal circumference was used, with the addition of a conjunctival flap. The size of the incision was adapted to the type of cataract to be delivered. The section followed the limbus as closely as possible, with a conjunctival flap of moderate size. Two silk sutures were inserted immediately after the section and drawn aside. Usually two or three more sutures, sometimes four, were added after the end of the operation. The sutures were of the straight through and through type, of fine braided silk (Deknatel N004 B 1 test) tied in three knots. Carrel arterial needles, size B 21, full curved, were used for the conjunctival suturing.

A small iridectomy was done in most cases. In cases in which a simple extraction was performed a buttonhole peripheral iridectomy was done before delivery of the lens.

Extraction of the Lens.—The capsule of the lens was grasped at the junction of the lower and the middle third. Elschnig's or Arruga's forceps was used in most of the first 100 cases and my forceps in the remainder.

After the capsule was grasped, the forceps was held as steady as possible, with no rocking movement. Counterpressure was applied at the limbus below with the edge of the ordinary cataract spoon. The pressure was directed backward toward the optic nerve and was gentle and in no way comparable to the force formerly exerted in the Smith operation. Slight motions sidewise with the spoon to either side of 6 o'clock were made directly backward until the zonule ruptured and the lower edge of the lens appeared to luxate forward, after which the pressure was upward and a little backward, the forceps being synchronously withdrawn as the lens advanced upward through the wound. The forceps was never deliberately pulled on, except as pressure was applied below, and it was then withdrawn as the lens advanced upward through the incision.[3] The lens tumbled in most instances, though frequently it was delivered head on, depending on the nature and size of the nucleus. The forceps aided in bringing the lens through the wound and also in breaking the upper zonular fibers. Gradle[4] spoke of his method of intracapsular delivery as 80 per cent push and 20 per cent pull. The procedure described here might be considered as 95 per cent push and 5 per cent pull. After delivery, the sutures were immediately tied and additional sutures inserted. The pillars of the iris were then replaced.

If the capsule ruptured coming through the wound and there was a question of some remaining, the capsule forceps was again introduced just inside the lips of the wound and any remaining capsule was withdrawn. If the capsule ruptured before the lens was delivered, an ordinary extracapsular procedure was carried out, after it was ascertained that a large portion of the capsule had been removed.

A 2 per cent solution of pilocarpine nitrate was instilled into the eye in all cases at the close of the operation, a 1 per cent solution of physostigmine salicylate being added if the simple extraction was used. An ointment containing mercury bichloride in a concentration of 1:5,000 was placed between the lids

3. This method of delivery of the lens was first demonstrated to me by Dr. William L. Benedict. I have been unable to find any published description of the exact technic employed, though it corresponds closely to that described by Lancaster in his discussion before this society in 1933.

4. Gradle, H.: Intracapsular Versus Extracapsular Extraction of Cataract, Tr. Sect. Ophth., A. M. A., 1933, p. 186.

and especially over the lacrimal puncta. Gauze patches treated with petrolatum were placed over the lids, and the eyes were lightly bandaged for four days.

Postoperative Treatment.—The first dressing was done on the fourth day in cases in which complete iridectomy had been performed and after forty-eight hours in cases in which simple extraction was done. The patients were kept in bed from four to five days, the use of a back rest being permitted after forty-eight hours. A soapsuds enema was given on the fourth day. Sutures were removed on the seventh day unless they spontaneously extruded. The eye which was not operated on was uncovered on the fourth day after operation, and the eye subjected to operation was dressed on alternate days until the time of discharge, which for private patients was usually the tenth or twelfth day. The average stay in the hospital for patients in the wards was fourteen days. A 1 per cent solution of atropine was used at the postoperative dressings if needed, but frequently only one or two instillations were required, since the reaction was usually slight after the intracapsular procedure.

Refraction was done on the patients in the wards at the end of two weeks for a record of the visual acuity, but glasses were not prescribed until six weeks later. The records show that a number of patients failed to return to the hospital for final refraction; so the vision recorded is that at the time of discharge. The records show that vision was usually improved at the end of six weeks.

STATISTICAL ANALYSIS OF TWO HUNDRED INTRACAPSULAR EXTRACTIONS

Age.—The ages of the patients are shown in table 1, the grouping being done in decades from 40 to 90. (The youngest patient operated on was 42, and the oldest, 87.)

Description of Cataracts.—The state of maturity of the cataracts in this series is shown in table 2. One hundred and twenty-five of the cataracts were classed as mature, 58 as immature and 17 as hypermature. I have found it profitable to study each lens carefully after removal by tearing off the capsule and examining the cortex and nucleus. In this way one increases one's ability to judge more exactly the nature of the cataract before operation and thereby to choose the proper procedure and estimate the size of the section for removal in a given case.

With regard to the primary cause of the cataracts, I am unable to offer any important evidence. Nineteen patients had diabetes in varying states, though only 6 of these showed diabetic retinitis and only 2 were considered to have diabetic cataracts. Two patients had a high degree of myopia. Such patients are usually operated on by the extracapsular procedure, but owing to the marked immaturity of the cataracts of these 2 patients the intracapsular method was employed. Five patients had chronic simple glaucoma, in 1 of whom the eye had been trephined ten years earlier, with complete control of the tension. In this patient's case, incidentally, the cataract was extracted with complete success, with the capsule intact and with no loss of vitreous. There was 1 patient with an allergic cataract. In 1 case a detachment of the retina had existed for probably eight years. A presumptive preoperative diag-

nosis of detachment or intraocular tumor was made, and an extensive detachment was observed on the operating table immediately after removal of the lens, discernible by oblique illumination. In several cases there was subluxation of hypermature cataracts, with vitreous in the anterior chamber; these will be referred to later. In 1 case there had been an old mild plastic iritis, with slight posterior synechia.

Method of Operation.—Iridectomy, either preliminary or at the time of extraction, was performed in most cases, as shown in table 3. Personally, unless there is some definite indication to the contrary, I prefer the combined extraction, but others in the ophthalmic service prefer preliminary iridectomy. There is no occasion to discuss the rela-

TABLE 1.—*Ages of Two Hundred Patients Subjected to Intracapsular Cataract Extraction*

Age	Number of Patients
40 to 50 years	10
50 to 60 years	34
60 to 70 years	65
70 to 80 years	82
80 to 90 years	9
	200
Sex	
Men	109
Women	91
	200

TABLE 2.—*Description of Cataracts*

Type of Cataract		Number
Mature		125
Sclerosed	58	
Ordinary senile	67	
Immature		58
Amber nucleus, clear cortex	21	
Ordinary immature, including postsubcapsular	37	
Hypermature		17
Shrunken	15	
Fluid cortex	1	
Rubbery capsule	1	
		200

tive merits of this practice here, as this is too well known and various surgeons differ in their preference. The simple extraction gives the ideal postoperative result when successfully performed, but from the standpoint of safety and simplicity of extraction complete iridectomy has a definite advantage. The 10 patients on whom simple extraction was done were younger subjects, and the results were highly successful from an operative and a visual standpoint, though prolapse under the conjunctival flap necessitated excision in 1 case.

The cases in which intracapsular extraction was attempted but failed are not included in this group, as the operations fall in the class of

extracapsular extractions. Records have been accurately kept, however, of the course in these cases. We were successful in extracting 79 per cent of the cataracts in capsule.

Condition of Capsule on Delivery.—Rupture of the capsule in my hands has been most common in cases of completely sclerosed cataract, in which the lens is large, hard and brown and contains no subcapsular cortical material. The capsule appears thin and friable and does not move freely on the underlying cortex, as seen in many lenses, since none is present. Tumbling is difficult, owing to the fact the lens will not fold. Fortunately these cataracts are ideal for extracapsular delivery, owing to the absence of soft cortex, though I usually attempt to remove them in capsule. In cases of glistening, slightly intumescent cataract, especially when the capsule is under slight tension, the capsule ruptures easily when grasped with the forceps.

TABLE 3.—*Method of Operation*

Type of Operation	Number of Cases
Preliminary iridectomy	103
Combined extraction	87
Simple extraction with peripheral iridectomy	10
	200

TABLE 4.—*Condition of Capsule on Delivery*

Condition of Capsule	Number of Cases
Capsule intact	164 (+ cases), 82%
Lens + capsule (capsule broke coming through wound but was completely removed)	36 (+ — cases), 18%

The hypermature cataract dislocates most easily, though when it is of the morgagnian variety and swollen and distended, with a rubbery capsule, it is practically impossible to grasp it, even with Fuch's toothed forceps. I have removed some of these easily in capsule by external pressure only.

The immature cataract, with a clear cortex and amber nuclear haze, is ideal for extraction. The capsule moves freely on the underlying cortex, which one may demonstrate by rubbing the extracted lens between the thumb and the forefinger. The capsule therefore is easily grasped without rupturing, owing to this freedom of movement and to the presence of ample pultaceous subcapsular material. The loose capsule can be seen projecting up through the jaws of the forceps. This feature of the forceps which I designed was described at a meeting of the American Ophthalmological Society,[5] where it was exhibited. The

5. Davis, F. A.: Capsule Forceps for Intracapsular Cataract Extraction, Tr. Am. Ophth. Soc. **34**:239, 1936.

large area of capsule which can be grasped distributes the traction over a good portion of the equator of the lens.

The fully mature, homogeneous, grayish white cataract is usually easily delivered, the capsule holding well.

As a general rule, the operation is most successful on persons in the age group from 60 to 90 years, since the zonule appears weakened and breaks easily. One is surprised at times, however, by the ease of extraction of the soft, white, rapidly developing cataract which is seen in the age group from 40 to 50 years, if time is allowed for a full, deep chamber.

Prolapse of the Vitreous.—A record of cases in which vitreous was lost, with the last recorded vision, is shown in table 5. The vitreous appeared in the lips of the wound or was lost in varying amounts in

TABLE 5.—*Analysis of Cases on Basis of Loss of Vitreous*

Case No.	Amount of Vitreous Lost	Vision	Condition of Capsule
1	Small amount with delivery	20/15	Intact
2	Fluid vitreous; small amount flowed out during operation	20/20	Intact
3	Weak zonule; very little loss; lens fell back on grasping; lifted out in capsule	20/15	Intact
4	Tiniest amount; shrunken lens (15 years' duration) lifted out with forceps in capsule	20/200	Intact
5	Very small amount after delivery; poor cooperation	20/20	Intact
6	Moderate amount; lens fell into vitreous; delivered with loop	20/30	Intact
7	Tiniest bead; appeared after suturing wound on replacing pillar	20/30+	+—
8	Moderate loss after delivery	20/30	Intact
9	Four to five drops after delivery	20/20	Intact
10	Tiny drop after delivery	20/30	Intact
11	One drop after delivery	20/40	Intact
12	Two drops after delivery	20/30	Intact

12 cases, or in 6 per cent of the total number in which operation was performed. This number is less than that in another series of 100 cases in which extracapsular extraction was performed by substantially the same group of operators (aside from changing house officers) during the same period. In the latter series there was a loss of vitreous in 9 per cent of the cases. While this is contrary to the usual experience, Gradle [4] reported similar results in 200 cases in which operation was performed (intracapsular in 100 and extracapsular in 100) by him. His records show loss of vitreous in 3 per cent of the cases in which intracapsular extraction was performed and in 7 per cent of those in which extracapsular extraction was done, though there were but 80 intracapsular deliveries.

In table 5 it will be noted that loss of vitreous was followed by poor vision in but 2 cases, namely, cases 4 and 11. In case 4 the amount of vitreous lost was insignificant, and the lowered acuity of vision could not be attributed to this, though the exact cause was difficult to determine, since the retina was essentially normal. The cataract was of the

hypermature, shrunken type, of fifteen years' duration; the iris was tremulous, and the vitreous was degenerated though not fluid. The lens fell back slightly when the capsule forceps was applied. Delivery was easily accomplished by reintroduction of the forceps, and after the capsule was again grasped the lens was lifted out intact. No loop was used, nor was pressure exerted on the globe with the spoon. This method of delivery of a subluxated lens has not been sufficiently stressed. It is far superior to the older method of loop and spoon delivery, in which a considerable amount of vitreous may be lost.

In case 11 a drop of vitreous was lost. The healing was prompt, but according to the record vision was only 20/40 on the fourteenth day, when the patient was discharged. The patient never returned for final refraction.

If the zonule does not break readily with moderate pressure on the globe, we usually abandon the effort and remove the capsule with the Fuchs capsule forceps.

With the possible exception of some of the house officers' first operations, in which loss of vitreous was due to inexperience, vitreous seemed to appear without apparent cause and when least expected. My own experience has been similar in cases in which the extracapsular method was used. Vitreous may appear when there has been a minimum amount of trauma, occasionally after the section and at times on replacing a pillar, or when the patient suddenly moves the eye. It would seem that it must be related to the condition of the hyaloid and the consistency or condition of the vitreous itself. In other words, some patients seem predestined to this complication, while every surgeon is familiar with types of cases in which excessive manipulation and unusual trauma are not followed by prolapse. Unquestionably the thorough paralysis of the orbicularis muscle by the injection of procaine hydrochloride has reduced this hazard by the complete elimination of squeezing, and large losses of vitreous seem to have vanished. In my earlier cases avertin was used when it was suspected that the patient might be a "squeezer" or a "bad actor," but with increasing confidence in the operation and a more thorough akinesia I have found its use less important.

It is, of course, unnecessary to stress the importance of measuring intraocular tension with the tonometer in every case prior to operation. The intracapsular procedure is usually avoided if tension cannot be brought well within normal limits.

Prolapse of the Iris.—This complication has occurred in a limited number of cases in this series, as is shown in table 6.

As noted in the table (case 1), prolapse of one pillar occurred on the eleventh day. The patient had had an exceptionally quiet eye and

was dismissed on the tenth day. Too great activity bending over caused one pillar to prolapse through the wound, without loss of the anterior chamber. It was excised, with prompt recovery.

In case 2, in which simple extraction was performed, a prolapse, completely covered by the conjunctival flap, was found at the first dressing. The patient vomited several times during the first night after operation and during the next day, from no apparent cause, as the operation was performed under local anesthesia. Excision was followed by prompt recovery, with little reaction and excellent vision.

In case 3 prolapse of one pillar occurred when the patient bumped her eye.

In case 4 there was hemorrhage into the anterior chamber, and the wound was sprung, with incarceration of one pillar beneath the flap, excision being necessary.

In cases 5, 6, 7 and 8 one pillar caught in the wound, but no intervention was necessary, since the eyes were quiet.

TABLE 6.—*Analysis of Cases on Basis of Incarceration and Prolapse of the Iris*

Case No.	Condition of Iris
1	Prolapse of one pillar on eleventh day (overactivity at home)
2	Prolapse after simple extraction (postoperative vomiting)
3	Prolapse of one pillar (patient struck eye)
4	Prolapse of one pillar (hemorrhage in the anterior chamber; wound sprung)
5	Caught pillar; no prolapse
6	Caught pillar; no prolapse
7	Caught pillar; no prolapse
8	Caught pillar; no prolapse

Hemorrhage into Anterior Chamber.—Hemorrhage into the anterior chamber was observed at various dates during the postoperative period from the fourth to the fourteenth day. In some of the cases its occurrence was definitely associated with trauma, such as turning in bed with pressure on the eye, getting out of bed and falling to the floor and on straining from an enema, but there were a number of cases in which hemorrhage seemed to occur spontaneously without injury of any kind. Most of the hemorrhages were small and cleared up promptly, but several were of considerable amount, completely filling the anterior chamber and in 2 cases causing a bulging of the flap with external oozing. The relative frequency of this mishap is puzzling aside from those hemorrhages directly due to trauma. Wright, in his masterly series of lectures on cataract, appearing in the *American Journal of Ophthalmology,* stated in his third lecture[6] that he has encountered this accident in over 10 per cent of his cases and that in some years it

6. Wright, R. E.: Lectures on Cataract: Posterior-Segment Complications in the Postoperative Period; Some Difficult Extractions, Am. J. Ophth. **20**:376 (April) 1937.

has been as high as 20 per cent. No better description of the varied aspects of this complication can be found anywhere. Detailed analysis of our cases in respect to hemorrhage will be found in table 7.

In this table it is revealed that postoperative hemorrhages into the anterior chamber occurred in 12 cases, in 1 of which it invaded the vitreous, and that quiet hemorrhages into the vitreous occurred in 2 cases. These results are surprisingly similar to the average of 7.6 per cent reported by Vail [7] in statistics gathered from various sources and presented in his thesis, "Hyphemia After Cataract Extraction. My experience also confirms this writer's conclusion that the source of the bleeding in most cases seems most likely from the wound rather than from the iris.

TABLE 7.—*Analysis of Cases on Basis of Postoperative Hemorrhage into the Anterior Chamber*

Case No.	Day	Description of Hemorrhage	Cause of Hemorrhage	Final Vision
1	9th	Into the anterior chamber	Unknown	20/15
2	5th	Into the anterior chamber	Unknown	20/15
3	6th	Into the anterior chamber	Unknown	20/20
4	6th	Very slight; into the anterior chamber	Unknown	20/20
5	6th	Into the anterior chamber	Patient rolled onto eye	20/15
6	6th	Into the anterior chamber	Patient tore off bandage; coughing uncontrollable (same for other eye, extracapsular extraction)	20/40
7	6th	Into the anterior chamber	Unknown	20/15
8	5th	Severe; into the vitreous and anterior chamber	Patient bumped eye; prolapsed iris excised	Hand movements
9	4th	Small; into the anterior chamber	Unknown	20/20
10	4th	Small; into the anterior chamber	Fluid vitreous; no loss	20/15
11	17th	Small; into the anterior chamber	Patient struck eye	20/20
12	6th	Into the anterior chamber; quiet hemorrhage into the vitreous	Unknown (similar hyphema in other eye 6 mo. later)	20/30
13	8th	Into the anterior chamber	Loss of vitreous at operation; blood remained 5 wk.	20/30
14	?	Quiet hemorrhage into the vitreous	Complicated cataract; fluid vitreous	20/30

In my experience postoperative hemorrhage occurred as frequently in cases in which the extracapsular method was used; so it does not appear to be due to the intracapsular procedure. Hemorrhage was not definitely associated with diabetes in this series of cases, though I have observed a number of such cases previously in which the extracapsular method was used in which hemorrhage seemed to be related to a diabetic condition.

This study reveals no definite cause for the bleeding aside from postoperative trauma. A poor section, too scleral, appears definitely to predispose to postoperative hemorrhage. None occurred after simple

7. Vail, D. T., Jr.: Hyphemia After Cataract Extraction, Tr. Am. Ophth. Soc. **31**:496, 1933.

extractions. Quiet hemorrhage into the vitreous occurred in 2 cases; the hemorrhages were discovered only at the end of two weeks, when refraction and ophthalmoscopic examination were undertaken. The hemorrhages cleared up satisfactorily, though vitreous streamers remained. In only 1 case (8) did hemorrhage result in a marked reduction in vision, and this was a postoperative hemorrhage into the anterior chamber and vitreous on the fifth day, due to trauma.

Visual Results.—The visual results are presented in table 8. It should be noted that most of the patients operated on come from distant parts of the state, so that in many instances the vision recorded was that determined after a brief refraction on the day of discharge, usually the fourteenth day. The final refraction for some of these patients was done by their local physicians, though an effort was made to have all return for a thorough refraction and glasses at the end of from six weeks to two months.

TABLE 8.—*Record of Visual Results*

Vision	No. of Cases	Percentage
20/15	82	83% (20/30 or better)
20/20	54	
20/30	30	
20/40	8	
20/50	7	
20/70	5	
20/100	5	17% (20/40 or less)
20/200	6	
Hand movements	2	
Unknown	1	

As shown in table 8, 83 per cent of the patients obtained vision of 20/30 or better, while 17 per cent obtained vision of 20/40 or less. Vision of 20/30 or better was obtained in 71 per cent of the series of 100 cases in which extracapsular extraction was done.

In table 9 will be found an analysis of the cases in which intracapsular extraction resulted in vision of 20/40 or less. Eight of the 34 patients failed to return for final refraction, and the visual results in these cases are those obtained on the day of discharge, usually the fourteenth day.

Lowered visual acuity from loss of vitreous could not have been a contributing factor in but 2 cases in this group, and in 1 of these the visual record of 20/40 was that on the date of discharge—the fourteenth day. One patient with vision of 20/200 had a hypermature, shrunken cataract, of fifteen years' duration, with vitreous in the anterior chamber. The retina appeared normal, and the loss of vitreous was slight. Vision, however, could not be improved.

Aside from these cases and 1 in which hemorrhage occurred into the anterior chamber and vitreous, lowered visual acuity was chiefly due

to preexisting disease, such as a high degree of myopia, chronic glaucoma, diabetic retinitis, retinitis pigmentosa and macular degeneration. In 1 case both eyes were operated on, with perfect postoperative results, but vision of only 20/200 was obtained. The eyes appeared normal. The patient spoke no English and was of extremely low mentality.

Miscellaneous Data.—A few other features deserve comment.

In 1 patient aged 82 scarlet fever and otitis media developed on the sixth day after operation, with no interruption or complication in the

TABLE 9.—*Analysis of Cases in Which Vision of Less Than 20/30 Was Obtained*

Degree of Vision	Contributing Factors	No. of Patients
Vision 20/40		
	Patient did not return for refraction (vision on discharge)	3
	Myopia	1
	Chronic glaucoma, preoperative	1
	Hyphemia (patient tore off bandages)	1
	Hypermature cataract	1
	Cause unknown	1
Vision 20/50		
	Diabetic retinitis	3
	Chronic glaucoma; subluxated lens	1
	Macular degeneration	1
	Patient did not return for refraction (vision on 14th day)	2
Vision 20/70		
	Retinal hemorrhages; hypertensive retinitis	1
	Hypermature cataract (15 years' duration); squint	1
	Macular degeneration	1
	Patient did not return for refraction (vision on 14th day)	2
Vision 20/100		
	Corneal dystrophy	1
	Hypermature cataract (15 years' duration)	1
	Diabetic retinitis	1
	High degree of myopia (20 D.); squint; vitreous change	1
	Patient did not return for refraction (vision on 14th day)	1
Vision 20/200		
	Vitreous into anterior chamber; slight loss; complicated cataract (15 years' duration	1
	Diabetic retinitis	2
	Retinal detachment (8 years preoperatively; observed on operating table)	1
	Unknown (perfect intracapsular operation; normal fundi)	2
Vision less than 20/200		
	Retinitis pigmentosa	1
	Hemorrhages into vitreous and anterior chamber 5th postoperative day	1
Vision unknown		
	Death from pneumonia (7th postoperative day)	1

healing process, and vision of 20/20 was obtained. One other patient had mild otitis media. Six patients were disoriented, and 2 might be classed as having postoperative mania. All became rational promptly on removal of the patch from the eye which was not operated on and on getting out of bed. Several patients had delayed restoration of the anterior chamber, some as late as the ninth day. Watchful waiting, rest in bed and the use of a 2 per cent solution of pilocarpine nitrate daily was the only treatment employed, and finally the wound healed

in all cases without further complication. Late bulging of the wound under the flap occurred in a few cases, but there was never actual prolapse of vitreous. External oozing occurred in 2 of the cases with hyphemia. True iritis was observed in but 3 cases, and in none of them was it severe. In view of the statements of Knapp [8] concerning this complication, I have searched our records for cases which might come in this category but can find only 3 which were so classified. The fact that fourteen days was the average length of hospitalization corroborates this in a way, since all patients were kept in the hospital until their eyes were quiet. In some cases the period of hospitalization was five weeks, but for the most part this was in cases in which hemorrhage had occurred. No doubt some degree of irritation of the iris was present in some of the cases, but frank iritis was exceptional.

Postoperative vomiting occurred infrequently—occasionally when no postoperative sedative was used. We scrupulously avoid the postoperative use of morphine, though it is used with scopolamine hydrobromide, 1/200 grain (0.00032 Gm.), as a preliminary sedative in cases in which operation is done with avertin anesthesia. One-half grain (0.03 Gm.) of codeine sulfate and 5 grains (0.32 Gm.) of acetylsalicylic acid is the only postoperative sedative used, and this is avoided when possible.

Slit lamp study was carried out on all patients returning for refraction after six weeks. The anterior surface of the vitreous occasionally showed a bulging forward through the pupillary area, and rarely a rupture of the hyaloid was seen. At times holes were seen in the hyaloid, and pigment dust from the iris or blood was occasionally observed. As a rule, the hyaloid appeared smooth and flat. Opacities of the vitreous and fine streamers were seen in a number of cases, though no statistical study has been made of these. This condition is seen in so many cases before operation that it would be difficult to obtain an accurate record.

The number of patients in whom hypertensive cardiovascular disease was recorded was large. This condition rarely appeared to interfere with a satisfactory postoperative result.

Records of astigmatism conform so closely to the many observations recorded by others that no especial comment regarding this seems indicated.

In this series it will be noted there was not a single postoperative intraocular infection and no expulsive subchoroidal hemorrhage. Likewise, to date there has been no postoperative detachment of the retina.

8. Knapp, A.: Complications of the Forceps Intracapsular Cataract Operation, Tr. Am. Ophth. Soc. **34**:162, 1936.

Choroidal detachment has been observed in only 1 case, but patients are not examined with the ophthalmoscope until the fourteenth day, except in rare instances.

COMMENT

After the warnings of such an outstanding authority as Wright,[6] one hesitates to make any dogmatic statements concerning an operation when the series reported is not large. When one has employed the extracapsular operation exclusively for nineteen years, however, and then adds the intracapsular procedure for a period of four years, it is difficult to refrain from making comparison.

One of the greatest advantages of the intracapsular operation is the freedom it permits in operating on the immature cataract. Long waiting in semiblindness can be avoided in most cases. The cataract with light nuclear sclerosis and a clear peripheral cortex is ideal for the intracapsular procedure, and the operation can be undertaken as soon as the anterior chamber is of sufficient depth to permit a satisfactory section. The actual technic of the intracapsular procedure is simpler than that of the extracapsular procedure, if one uses forceps for removal of the anterior capsule in the latter operation, since various manipulations incident to the removal of retained cortex, such as massage of the cornea and irrigation of the anterior chamber, are completely eliminated. Careless irrigation with improper solutions has not infrequently resulted in marked postoperative irritation and at times permanent clouding of the cornea.

Naturally one must practice the technic of intracapsular removal a sufficient number of times to become thoroughly familiar with the operation. Coordination between pressure and light traction must be developed, and fear of sudden loss of vitreous must be overcome.

Fewer instruments are introduced into the anterior chamber, and the incidence of infection should be lower. Usually the stay in the hospital has been definitely shortened. Postoperative complications have been reduced in this series as compared with the cases in which extracapsular extraction was performed. The patients on the whole have been much happier owing to the freedom from postoperative irritation and the shorter period of hospitalization. No discissions for after-cataract are required, with their attendant embarrassment.

I believe that in the average case in which there are no complications the intracapsular operation, with some form of forceps extraction, should be the operation of choice. The optimum achievement in cataract extraction is obtained by a successful intracapsular procedure, with the preservation of a round pupil. The feature of safety, however, must always be kept in mind, and unquestionably some form of complete iridectomy adds to this, as stressed by Knapp and others. I believe that iridectomy should always be used for elderly persons, who poten-

tially are not cooperative and for whom a cosmetic result is not so essential. I prefer the simple extraction with peripheral buttonhole iridectomy for younger subjects. With a combined or preliminary iridectomy, as one prefers, the intracapsular operation appears as safe as the extracapsular procedure when there are no complications. If one considers the possible postoperative complications of the capsulotomy operation, the intracapsular procedure, in my opinion, is the safer.

Peters,[9] in discussing Knapp's [10] paper on his third series of 100 intracapsular extractions, stated that "if we eliminate the question of surgical skill and compare the results of the intra- and extracapsular methods there can be no reason for difference of opinion as to the much greater value of the intracapsular extraction."

Knapp [10] pointed out that incarceration of the iris, opacities of the vitreous after removal of the cataract and glaucoma are all reduced to a minimum, while recovery is more rapid.

Török [11] in 1916, discussing his method of forceps extraction, stated that it is simple and easy for any one who is experienced with the ordinary Graefe extraction and, further, that the danger of loss of vitreous is even less, because pressure is less when traction is used.

Contrary to the usual teaching and belief with regard to the greater hazard of loss of vitreous with the intracapsular operation, I have grown less apprehensive about this phase of the procedure, as the number of my operations has increased, and, barring the exceptional case, it seems to me this complication should be no more frequently incurred than with the extracapsular method.

The outstanding features of the uncomplicated intracapsular extraction are the simplicity of the operative procedure, the slight postoperative reaction in most cases, the shortened period of postoperative healing and the rapid restoration of excellent vision.

Every ophthalmologist who removes any number of cataracts should be familiar with some form of intracapsular extraction. It may never completely replace the capsulotomy procedure, but certain types of cataract are best suited to the intracapsular method, and those who do not practice both methods will find themselves handicapped in the management of many of their patients.

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9. Peters, in discussion on Knapp.[10]

10. Knapp, A.: Extraction of Cataract: Report of a Third Hundred Successive Extractions in the Capsule After Preliminary Subluxation with the Capsule Forceps, Arch. Ophth. **5**:575 (April) 1931.

11. Török, E.: Extraction of Cataracts, Ann. Ophth. **25**:712 (Oct.) 1916.

PRACTICE OF DARK ADAPTATION

A REVIEW

JACOB B. FELDMAN, M.D.
PHILADELPHIA

The purpose of this paper is to review the subject of dark adaptation and the technic for its study. A brief sketch of the common instruments used to record dark adaptation is given, with particular reference to the method by which the quality of the light is retained throughout its entire varying range of intensity. The quantitative instrument I now use in my studies is described.

Dark adaptation, as the term implies, is the ability which the eye has to adapt itself to darkness. For example, if one enters a darkened cinema theater on a sunny day, it is impossible at first to see any seats, but after a specified number of minutes objects are somewhat clearer. As time goes on, improvement in vision is noted, until the sight becomes as distinct as if the theater were illuminated. This physiologic function of the normal human being is one of dark adaptation. It takes a definite time for the eyes to become adapted to dark. Lack of perfection of this visibility in darkness or slowness of the eyes to gain clear vision constitutes pathologic dark adaptation, or dysaptation.[1]

PHYSIOLOGY OF ADAPTATION

The eye is capable of two types of vision: (1) form and color sense, for which good light is necessary, and (2) light sense, which is detected in subdued light or twilight. Form and color sense develops at about the sixth month of life. Light sense, however, is possibly fully developed soon after birth.

Believers in the von Kries[2] theory give credit to the cones of the retina for daylight vision (form and color sense). According to this theory, the rods, aided by the visual purple, are responsible for vision in the dark. The rods are more responsive to rays of the shorter wavelengths.

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1. Dysaptation is the name given to a pathologic or abnormally high light threshold reading by C. Edmund and S. Clemmesen (On Deficiency of A Vitamin and Visual Dysaptation, Copenhagen, Levin & Munksgaard, 1936).

2. Only the von Kries theory is discussed here because I feel that this theory more adequately explains dark adaptation than any of the others. The various theories and their evaluation can be found in standard works on physiology.

Opponents of the duplicity theory of von Kries refute the dual action of rods and cones. They say that there is equality in sensitivity between the rods and the cones after prolonged dark adaptation. They are of the opinion that photopic and scotopic vision is a function of the cones which utilizes visual purple.

Believers in the von Kries theory, however, to prove their contention, note that nocturnal, or night-prowling, animals, such as moles, hedgehogs, mice, rats, night apes, owls and geckos, possess an abundance of rods, while animals which forage for food and carry on during daylight, such as the chameleon, birds, lizards and some reptiles, have an excess of cones in the retina.

Regardless of one's opinion relative to the von Kries theory, it is certain that the retina responds to two types of vision: (1) daylight, or photopic vision and (2) twilight, or scotopic vision. The latter is accomplished through the aid of the visual purple.

The changes in photopic vision are so rapid and so variable that they cannot be studied accurately.

Scotopic vision, on the other hand, is slower and can be investigated. This is accomplished by a study of the dark adaptation.

VITAMIN A

Studies have shown that there is a relation between visual purple and vitamin A and also between A avitaminosis and certain ocular diseases.

Wolff [3] and Moore [4] have found the liver to be the main source of vitamin A in the body.

Tansley [5] has been able to show by photography the quantitative differences of regeneration of the visual purple in normal rats and in rats deficient in vitamin A.

Wald, according to Matthews,[6] accounted for the presence of vitamin A in the retinal tissue by the combining of vitamin A in the blood with the visual purple in the eye.

Tassman [7] has reviewed the subject of vitamin A as related to ophthalmology.

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3. Wolff, L. K.: On the Quantity of Vitamin A Present in the Human Liver, Lancet **2**:617, 1932.

4. Moore, T.: Vitamin A Reserves of the Human Liver in Health and Disease, Lancet **2**:669, 1932.

5. Tansley, K.: The Regeneration of Visual Purple: Its Relation to Dark Adaptation and Night Blindness, J. Physiol. **71**:442, 1931.

6. Matthews, A. P.: Principles of Biochemistry, Baltimore, William Wood & Company, 1936, p. 412.

7. Tassman, I. S.: Dietary Deficiency and Ocular Disease, Arch. Ophth. **8**: 580 (Oct.) 1932.

CLINICAL IMPORTANCE OF STUDIES OF DARK ADAPTATION

Dark adaptation is the measure of the function of the receiving apparatus. Pathologic dark adaptation, therefore, indicates interference with the function of the deeper structures of the retina, viz., the rods, choroid and the visual purple. Study of the dark adaptation of a patient with cataract would therefore be of great aid in the preoperative prognosis. A study of dark adaptation shortly after an operation for glaucoma would indicate the restoration of function in the deeper retinal structures.

In the absence of an ocular pathologic process, dysaptation of moderate degree would be strong presumptive evidence of a vitamin deficiency.

Avitaminosis A has been found to play an important role in such diseases as hepatic and intestinal disorders,[8] renal calculi [9] and certain diseases of the blood,[10] or of the skin.[11]

I have found some of these conditions to be accompanied with pathologic dark adaptation.[12]

Normal dark adaptation is important to safety in night travel in the air, on the sea and on the highway.[13] Many accidents are due to more or less pronounced night blindness, of which the driver may be unaware.

CLASSIFICATION OF CLINICAL CASES FOR STUDY OF DARK ADAPTATION

I divide all of my patients who are to be subjected to a study of dark adaptation into three arbitrary groups. There is no sharp demarcation in the classification. Examination of a patient may reveal a pathologic condition which will permit classification under more than one group.

Group 1.—In the first group are placed patients in whom a physical or an anatomic disturbance is found. Included here are patients with-

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8. Eusterman, G. B., and Wilbur, D. L.: Clinical Features of Vitamin A Deficiency, J. A. M. A. **98**:2054 (June 11) 1932.

9. Higgins, C. C.: The Experimental Production of Urinary Calculi, Urol. & Cutan. Rev. **38**:33, 1934.

10. Berglund, H.; Keefer, C. S., and Yang, C. S.: Deficiency Anemia in Chinese, Responding to Cod Liver Oil, Proc. Soc. Exper. Biol. & Med. **26**:418, 1929.

11. Frazier, C. N., and Hu, C. K.: Nature and Distribution According to Age of Cutaneous Manifestations of Vitamin A Deficiency, Arch. Dermat. & Syph. **33**:825 (May) 1936.

12. Feldman, J. B.: Further Studies on Dark Adaptation, Arch. Ophth. **17**:648 (April) 1937.

13. (*a*) Luckiesh, M., and Moss, F. K.: The Science of Seeing, New York, D. Van Nostrand Company, Inc., 1937, p. 22. (*b*) Jeghers, J.: Night Blindness Due to Vitamin A Deficiency: A Consideration of Its Importance in Traffic Problems, New England J. Med. **216**:51, 1937. (*c*) Feldman.[12]

out any visible retinal pathologic process or corneal opacity attributable to vitamin A deficiency. There is present, however, a physical disturbance in the size or function of the eye or a turbidity of the ocular media, which might be expected to cause pathologic dark adaptation. Included in this group are patients with conditions dependent on the factors of age and sex; patients with refractive errors of various sorts, and persons with color blindness, conical cornea, nystagmus, opacities of the cornea or of the vitreous, cataract, without other complications, dislocation of the lens and amblyopia ex anopsia.

Examination of a number of patients belonging to this group has shown normal dark adaptation.[14]

Group 2.—In the second group are placed patients in whom a physiologic disturbance is found. Here are included patients with a definite retinal pathologic process which is disturbing the physiologic function of the retina (rods). In this group are patients with choroiditis and retinal detachment; a great majority of patients with glaucoma and retinal arteriosclerosis; patients with congenital hemeralopia, such as retinitis pigmentosa and retinitis punctata albescens, and patients with idiopathic or acquired hemeralopia, such as the night blindness caused by overexposure to light, the type of night blindness seen in shell-shocked soldiers and a night blindness common to coal miners,[15] in whom there may be overactivity of the visual purple.

A pathologic dark adaptation of varying degrees, which is usually higher than that in patients in group 3, is found in all patients in this group.

Group 3.—In the third group are placed patients in whom a biochemical disturbance is found. This group includes persons in whom vitamin A was deficient or did not combine with the visual purple. Several manifestations of vitamin A deficiency may present themselves. Thus one encounters clinical avitaminosis A, with or without xerophthalmia. This entity may manifest itself as a disease of the mucosa of the respiratory tract, as some types of disease of the blood or of the skin or as renal calculi, jaundice or some hepatic dyscrasia. A study of the dark adaptation of such persons often gives a moderately pathologic dark adaptation graph.

HISTORY OF THE STUDY OF DARK ADAPTATION

A brief history of the beginnings of the study of dark adaptation would include the work of Aubert,[16] who in 1865, by using a heated

14. Feldman, J. B.: (*a*) Dark Adaptation as a Clinical Test, Arch. Ophth. **15**:1004 (June) 1936; (*b*) footnote 12.

15. Percival, A. S.: Light Sense, Tr. Ophth. Soc. U. Kingdom **40**:311, 1920.

16. Aubert, H.: Untersuchungen ueber die Sinnesfähigkeit der Netzhaut, Ann. d. Phys. u. Chem. **115**:87, 1862.

platinum wire as a light stimulus in a dark room, was the first to study systematically the minimum light visible, or the light threshold. Charpentier [17] in 1880 showed the value of previous light adaptation on the subsequent study of the dark adaptation. According to Hay,[18] Förster in 1873 constructed the first scientific instrument to determine the light threshold and the course of dark adaptation. Since these early dates the literature on the subject has been augmented by various studies made on different phases of dark adaptation with a compilation of the data by Adams.[19]

A review of the literature reveals descriptions of some of the better known instruments, the outstanding feature of each being the novel way by which the quality of the light can be controlled throughout an entire study.

1. According to Lythgoe,[20] Best used "radiolite" screens for test objects.

2. Derby and his co-workers [21] used a photometric wedge in a portable instrument or in a projection lantern.

3. Downey [22] used "marvelite" disks. These disks are made by impregnating zinc sulfide with radium, over which celluloid disks are placed.

4. Edmund and Clemmesen [23] used a modified Masson disk with Tscherning photometric glasses.

5. Ferree and Rand [24] used a projector, with an iris diaphragm to step down the illumination.

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17. Charpentier, A.: Sur les variations de la sensibilité lumineuse suivant l'étendue des parties rétiniennes excités, Compt. rend. Acad. d. sc. **91**:995, 1880.

18. Hay, P. J.: Studies in the Light Sense, Arch. Ophth. **39**:160, 1905.

19. Adams, D.: Dark Adaptation: A Review of the Literature, Medical Research Council, Special Report Series, no. 127, London, His Majesty's Stationery Office, 1929.

20. Lythgoe, R. J.: Illumination and Visual Capacities, Medical Research Council, Special Report Series, no. 104, London, His Majesty's Stationery Office, 1926, p. 19.

21. Derby, G. S.; Chandler, P. A., and Sloane, L. L.: A Portable Adaptometer, Tr. Am. Ophth. Soc. **27**:110, 1929.

22. Downey, J. W.: Determination of Minimum Light Sense and Retinal Dark Adaptation with Presentation of a New Type of Photometer, Arch. Ophth. **2**:13, 1912.

23. Edmund, C., and Clemmesen, S.: On Deficiency of A Vitamin and Visual Dysaptation, Copenhagen, Levin & Munksgaard, 1936.

24. Ferree, C. E., and Rand, G.: Lantern and Appartus for Testing the Light Sense and for Determining the Acuity at Low Illuminations, Am. J. Ophth. **3**:335, 1920.

6. Feldman [25] described an instrument containing a neutral filter and a constant light. With this instrument, if the patient recognizes within five minutes the light stimulus and the direction it takes, he is considered to have normal adaptation.

7. Hay [18] employed an adjustable diaphragm with two pairs of Nicol prisms, thus making use of a polarized light in his photometer.

8. Hecht [26] used a light in a movable carriage. The amount of light decreases as the carriage recedes.

9. In the Nagel adaptometer [27] an Aubert diaphragm is used for fine changes of brightness; three screens, each containing a number of holes, are used to reduce further the original amount of light filtered through three milk glass plates.

10. Percival [28] used a black ring on a gray disk, the disk being rotated.

11. The Piper modification [29] of the Nagel adaptometer contains two Aubert diaphragms in addition to the screens used by Nagel.

12. Spaeth [30] used a mask containing miniature five dice openings, with prisms. The mask is placed in front of the patient's eyes, making it possible to record the dark adaptation in the ordinary examining room.

13. Waite and his co-workers [31] called attention to Förster's instrument, which makes use of an adjustable diaphragm, and to Nuttings' apparatus, which makes use of a photometric wedge.

These various types of instruments should be sufficient to demonstrate the diverse methods used by various observers to give a range of light and the means by which this factor is controlled.

ESSENTIAL FACTORS IN RECORDING DARK ADAPTATION

The minimum light flux which the eye can see is a function of at least four variables which should be taken into consideration in studies of dark

25. Feldman, J. B.: Instrument for the Qualitative Study of Dark Adaptation, Arch. Ophth. **18**:821 (Nov.) 1937.

26. Hecht, S.: The Nature of Foveal Dark Adaptation, J. Gen. Physiol. **6**: 113, 1921.

27. Nagel, W. A.: Zwei Apparate für die augenärztliche Functionsprüfung, Ztschr. f. Augenh. **17**:201, 1907.

28. Percival, A. S.: Notes on Light Sense, Tr. Ophth. Soc. U. Kingdom **42**: 285, 1922.

29. Piper, H.: Zur messenden Untersuchungen und zur Theorie der Hell-Dunkel-Adaptation, Klin. Monatsbl. f. Augenh. **45**:357, 1907.

30. Spaeth, E. B.: Clinical Determination of the Light Threshold, Arch. Ophth. **11**:462 (March) 1934.

31. Waite, J. H.; Derby G. S., and Kirk, E. B.: Glaucoma, Tr. Ophth. Soc. U. Kingdom **45**:310, 1925.

adaptation [32] and particularly in the construction of an instrument. They are: (1) the size of light, (2) the brightness of light, (3) the surrounding field and (4) the duration or time the light stimulus acts on the retina.

In my instrument for a qualitative study of dark adaptation I used only the fourth factor as the variable (the duration of the light stimulus acting on the retina). The other three were constant. In the instrument for the quantitative test, which I am now using and which will be explained later, the size and surrounding field are the constants, and both the brightness and the duration of time the light stimulus acts on the retina are the variables.

Size of the Light.—In a study of dark adaptation the rods are principally investigated. Breuer and Pertz [33] have found in this work that the retina is most sensitive at the area of from 10 to 20 degrees from the fovea. The light stimulus of an adaptation apparatus should therefore include this area of the retina. The machine is turned on, and the patient indicates just when a light is seen. An added check on the veracity of the patient is to have him tell the direction which the light stimulus takes. This involves the use of a movable light stimulus, which is highly valuable, and which I have made use of in my qualitative device for dark adaptation study.

Ordinarily, I have found the temporohorizontal segment of the retina the easiest to study [13a] and indicative of the health of the balance of the retinal tissue in the majority of cases. This is the area usually studied by my larger apparatus for quantitative estimation of dark adaptation. When, however, a visible retinal pathologic process is present, the light stimulus may be placed in a position corresponding with the pathologic area of the retina which it is desirable to investigate.

Brightness of Light.—The instrument should have a wide range of light, with a gradual and constant increase in brightness. Most important, the quality of the light must remain the same throughout its entire range of intensity. When a rheostat is used in a dark adaptation machine, a pure white light of intense brilliancy becomes yellowish when the light is reduced. This reduction by a rheostat alters the quality of the light.

A device in the dark adaptation machine to retain its voltage regardless of whatever other electric machinery is used in the building is a

32. (*a*) Lythgoe, R. J.: Illumination and Visual Capacities, Medical Research Council, Special Report Series, no. 104, London, His Majesty's Stationery Office, 1926. (*b*) Cobb, P. W.: Light Sense, Am. J. Ophth. **15**:917, 1932.

33. Breuer, H., and Pertz, A.: Ueber die absolute Empfindlichkeit der verschiedenen Netzhautteile im dunkeladaptierten Auge, Ztschr. f. Psychol. u. Physiol. d. Sinnesorg. **15**:327, 1897.

worthy refinement. The lamp in the dark adaptation machine should be carefully selected and seasoned.

In the selection of a desirable instrument, the words of Lord Kelvin are to be considered: "If you can measure that of which you speak, and can express it by a number, you know something of your subject; but if you cannot measure it, your knowledge is meagre and unsatisfactory."

The light in the instrument selected should be calibrated. Usually this is done in millilamberts. Since this is essentially an ocular test, it is better to give the light threshold reading in photons.

A photon is a unit of retinal illumination. It is 1 candle per square meter per square millimeter of pupil.

The method of converting millilamberts into photons [34] is as follows: $B \times 10a/\pi = I$. B is the brightness in millilamberts; a is the area of the pupil; π is 3.142, and I is the illumination of the retina in photons.

Surrounding Field or Environment of the Eye.—The surrounding field should be one of contrast, such as, for example, the white stimulus on a dark background. This is easily obtained by doing the tests in an absolutely dark room.

Duration or Time the Light Stimulus Acts on the Retina.—In most dark adaptation tests this variable is the only one of the four which is used as the experimental variable, the remaining three being constant. An instrument utilizing "flicker" [35] will record in split seconds the time during which the light threshold readings are taken.

Other Features.—The ability to record in the dark the intensity of light threshold used during each test is another valuable feature of a dark adaptation machine.

Noiseless operation is an asset. The comfort of the patient by means of a suitable head and chin rest is to be considered and makes for more valid recordings.

It should always be remembered that the test for dark adaptation is at best a subjective one and that one is always at the tender mercy of the subject examined. The shortcomings are many if care is not utilized. The patient should always be made to understand in advance what is desired of him. Cooperation on the part of the subject is essential. Children and some adults are often apprehensive of an on-coming calamity when taking this test in the dark, or for some unknown reason they give inconsistent answers as to just when they

34. Troland, L. T.: Apparent Brightness: Its Conditions and Properties, Tr. Illum. Engin. Soc., 1916, p. 947.

35. Lythgoe, R. J., and Tansley, K.: The Adaptation of the Eye: Its Relation to the Critical Frequency of Flicker, Medical Research Council, Special Report Series, no. 134, London, His Majesty's Stationery Office, 1929.

see the light stimulus. Their fears are allayed and much time is saved by having several patients take the test together.

When the preexposure "light adaptation" is incorporated in the dark adaptation machine, more valid results are obtained. This will be explained under "Uniform Preexposure."

THE INSTRUMENT

The instrument I now use in the study of dark adaptation has been constructed on the principles which I have embodied in this paper, relative to the light flux, the comfort of the patient, the accuracy in controlling the quality of the light and the light threshold recordings. This instrument is depicted on figure 1, in which *A*, indicates the chin rest and forehead support; *B*, the light-adapting chamber, which can be pushed out of the way at swivel *C* (the patient's eyes can be seen while being adapted to light); *D*, the stand containing voltage control, assuring a constant voltage to the lamp in lamp house *E*, the lamp being carefully selected and seasoned; *F*, a thumb screw which alters and records the distance from the lamp to the filter *G*; *H*, a pin, one on each side of the light stimulus where the patient's finger is placed, indicating where the patient is to look; *I*, the self-recording table, divided by a pencil line for the right and left eye (the turning of the table alters the intensity of the uniform light only), and *J*, the light house for photometric measurement of the lamp in the lamp house.

Before any studies are made, a photometer inserted in opening *J* should register 22 foot candles when the lamp is placed 20 cm. from the filter by adjusting the thumb screw (*F*). This makes certain of the equality of the photometric value of the lamp for all tests made.

The lamp is carefully selected and seasoned, and the voltage to the lamp is constant. The quality of the light is constant throughout the entire range of illumination. The instrument is portable.

All these features assure the observer of an accurate, easily handled and easily controlled instrument.

TECHNIC OF DARK ADAPTATION STUDIES

Next in importance to a carefully constructed instrument is a uniform technic. In all my studies the following steps are carried out:

1. The history is recorded, and a complete ophthalmologic examination is made.
2. The size of the patient's pupil is regulated.
3. A uniform preexposure to light is used in every case.
4. The patient is told where to fix the eyes during the test.
5. A uniform thirty minute study is used for each eye, records being taken usually every three minutes.
6. A dark adaptation graph is plotted.

History and Ophthalmologic Examination.—Dark adaptation, according to Traquair,[36] is the response to the reaction of the receiving apparatus, i. e., the visual purple, the choroid and the bacillary layer of the retina. An ophthalmoscopic examination of each patient previous to studying the dark adaptation makes it possible for one to divide those with organic diseases (glaucoma, choroiditis and retinitis pigmentosa), in whom the ocular pathologic process is usually evident, from those

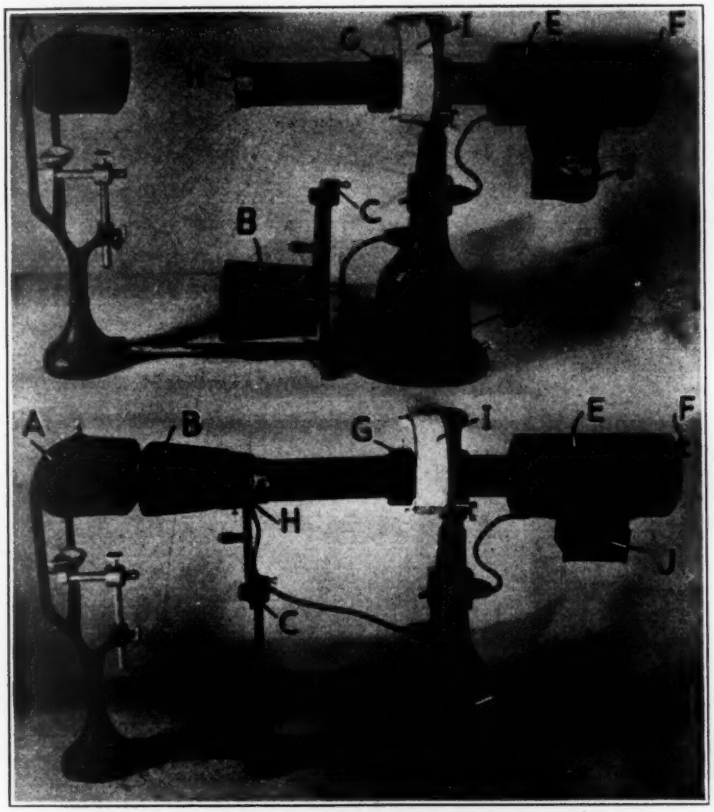

Fig. 1.—Machine for the study of dark adaptation.

with functional diseases (vitamin A deficiency), in whom the fundi are always normal. This aids in a correct classification of the graphs when they show dysaptation.

To give a concrete example: A. R., a 6 year old undernourished boy, was found to have choroiditis on ophthalmoscopic examination. If the ophthalmoscope had not revealed choroiditis, a diagnosis of dysaptation due to avitaminosis A might have been made from the appearance

36. Traquair, cited by Percival.[28]

of the child. The patient then would have been placed in group 3, whereas the correct classification is group 2.

Another case which shows the usefulness and importance of examinations of the fundi follows: R. S., a 60 year old man with renal calculi, was found on ophthalmoscopic examination to have retinal arteriosclerosis. Without the knowledge of the ophthalmoscopic findings, the patient would be placed in group 3 [37]; however, the proper classification is group 2.

Regulation of the Size of Patient's Pupils.—The size of the pupil plays an important role in the result obtained in taking the light threshold. In a patient with a previous iritis the synechia cut down the amount of light which strikes the retina. Even healthy pupils do not all dilate to the same extent in the dark room. When dark adaptation is recorded with the eye near the instrument, the accommodation necessary may slightly contract the pupil. Merrill and Oaks [38] have noted that the light sense is enhanced proportionally to the square root of the retinal area. Derby and his co-workers [39] have shown that an 8 mm. pupil admits sixteen times as much light as a 2 mm. pupil, thus inducing an error of 1,600 per cent if the pupillary size were not considered. They cited Møller as being the first of the Copenhagen group of investigators who took into account the size of the pupil as a factor in their investigation. Hecht,[26] recognizing the usefulness of knowledge of the size of the pupil, first used an artificial pupil, but finding this to be impractical he calculated the measurement of the pupil according to the formula of Reeves.[40]

Dilation of the pupil by a 1 per cent solution of atropine needs only casual mention. The increased size of the pupil necessitates a number of extra filters to step down the light for the light threshold reading. The use of a mydriatic may also subject the patient to glaucoma if there is any tendency to this condition. I have tried using an artificial pupil, but with little success. The slightest movement of the patient's eyes makes visibility impossible. The photographing of the pupil by infrared photography in the dark room was also abandoned. It is expensive, and sometimes a small amount of uveal pigment in the region of the pupil, which photographs black, gives the impression of a larger pupil. At best it does much to complicate a simple problem. The most effec-

37. Ezickson, W. J., and Feldman, J. B.: Signs of Vitamin A Deficiency in the Eye Correlated with Urinary Lithiasis, J. A. M. A. **109**:1706 (Nov. 20) 1937.

38. Merrill, H. G., and Oaks, L. W.: The Light Sense: Its Importance and Tests, Am. J. Opth. **9**:38, 1926.

39. Derby, G. S.; Chandler, P. A., and O'Brien, M. E.: Further Studies on the Light Sense in Early Glaucoma, Tr. Sect. Ophth., A. M. A., 1928, p. 37.

40. Reeves, P.: Rate of Pupillary Dilatation and Contraction, Psychol. Rev. **25**:330, 1918.

tive and easiest method consists of the use of a 1 or 2 per cent solution of pilocarpine nitrate to contract the pupil. Ordinarily, 1 drop every fifteen minutes for three doses is ample to contract the normal pupil to 1 mm. in size in the great number of cases. Sometimes I augment the pilocarpine by substituting a 1 per cent solution of physostigmine salicylate for the last instillation of pilocarpine. No untoward effect should follow this procedure. To date I have employed this method in about four thousand studies of dark adaptation, without any untoward effect. The method was first suggested by Derby and his co-workers and was used throughout all of their investigations. After contracting the pupil, Derby, following the suggestion of Reeves, calculated all data to a 5 mm. pupil for each eye examined. This was done so that the records of one patient could be easily compared with those of another. It was accomplished by multiplying the number of millilamberts obtained at the threshold reading by 2.50 for an 8 mm. pupil, by 1.33 for a 6 mm. pupil, by 1 for a 5 mm. pupil, by 0.66 for a 4 mm. pupil and by 0.16 for a 2 mm. pupil.

Uniform Preexposure.—Light adaptation, to precede the dark adaptation studies, is indicated to achieve more reliable results. In the beginning of this paper dark adaptation was explained by citing the inability of a person to see the seats in the theater for a short while after he came into the theater from the sunny street. The physiologic explanation of this phenomenon is that the sun bleached out the visual purple. When the person entered the dark theater, the vision improved in direct proportion to the regeneration of the visual purple.

In order to determine results which are consistent and which may be compared with the results of other workers, it is best to give all persons studied the same amount of preexposure to light.

Instead of preexposure of the eyes to light, the patient can be kept in a dark room for from one-half to one hour before dark adaptation studies are begun. This would, however, unduly prolong the examination.

Preexposure, or light adaptation, can also be accomplished by having the patient look at the clear blue sky on a bright sunny day. The shortcomings of preexposure to daylight are, of course, clearly evident when one considers the difficulty of light adaptation on a cloudy day or at night. A better and more accurate plan is to have the entire study made without the subject leaving his seat.

Lohman [41] calculated that two minutes is the exact time for an eye to become fully adapted to light. I have found this to be correct by practical experience, and to make certain still further that the light

41. Lohman, W.: Ueber Helladaptation, Ztschr. f. Psychol. u. Physiol. d. Sinnesorg. **41**:290, 1906.

adaptation is not undertimed I used three minutes in all cases. Some investigators use as high as ten minutes.

The instrument should have as brilliant and intense a light for preexposure as can be borne comfortably by the patient. A device in the apparatus making it possible to see if the subject is continually keeping the eyes open during preexposure is important. Often a patient will deliberately keep his eyes closed. The preexposure is for the purpose of bleaching out the visual purple. If this is not accomplished to the same extent and intensity for all patients, one cannot expect comparable data.

The change from preexposure (light adaptation) to dark adaptation should be sharp and clearcut.

Fixation of Eyes During the Dark Adaptation Test.—The question arises as to whether the patient should be told where to look when taking the dark adaptation test. It is the opinion of some observers that after fifteen minutes of dark adaptation the sensitivity of the rods and cones are equal and that the direction of gaze does not alter the threshold. Obviously, when only one reading is taken, and that before fifteen minutes, it is essential that the patient know where to look to see the light stimulus. On the other hand, when many readings are taken, directing the patient just where to look will yield a dark adaptation reading for the particular retinal area desired. Fixation may be accomplished by asking the patient to look at a minute red stimulus, of a wavelength of 6,500 angstroms or over.

An improvement over the red dot of light for fixation is to have the patient place the finger where it is desired that he fix his eyes. This involves the use of the muscle sense and makes the patient feel that he is taking an active and responsible part in the study.

Uniform Interval and Duration of Study of the Light Threshold.—The more often the dark adaptation readings are taken, the quicker the ultimate light threshold is obtained. In order that my graphs may be compared easily, I study each patient every three minutes for thirty minutes.

It is best to take the three minute readings of one eye and then the other, and so alternate, giving no preference to either one or the other eye.

It is worthy of note that summation does occur in dark adaptation; i. e., unlike cone threshold function, the minimum light threshold is lower when both eyes are used than when only one is tested. This fact is made use of in my qualitative device.[25]

The curves of sensitivity of the two eyes of a normal subject during dark adaptation are usually similar but not alway definitely the same.

Plotting of the Dark Adaptation Graph.[42]—Charpentier [17] and Jones [43] give twenty minutes as sufficient time for practical purposes for testing light sensitivity in a dark room. I have found sensitivity during dark adaptation tests up to thirty minutes. In some cases the sensitivity is definitely normal within twelve or fifteen minutes. When this occurs, it is not necessary to continue the readings. On occasion, I have studied the light sense of persons with pathologic conditions from one to four hours, at the end of which time the readings still continued to show pathologic dark adaptation. The readings of the first few three minute intervals are high, after which the sharp drop denoting the marked increase in sensitivity occurs. The eye of the great majority of normal persons becomes so sensitive to light that readings from then on are but a minute fraction of the original light just perceived by the patient.

The tabulations of the amount of light on my graphs are given as the ordinates, and the time of reading intervals, as the abscissas.

A series of dark adaptation readings for a normal person, when plotted on a graph, will produce a curve resembling a hyperbola. This hyperbola serves as a standard for the normal subject and enables one to compare the results obtained with those of other studies made by the same and other observers. Plotting a reading adds objective qualities to a subjective test. The observer can quickly recognize when the answer of the patient is incorrect. A sudden loss of sensitivity at one reading followed by a quick recovery on the following reading denotes inattention (fig. 2).

Just as in perimetry, characteristic changes in the visual field are associated with definite ocular or neuropathologic conditions, specific dark adaptation graphs have been associated with definite functional or organic pathologic involvement of the deeper retinal structures (rods, visual purple and choroid).

The following charts will, I believe, assist in illustrating the relative merits of the plotted graph over tabulated figures of light measurements in the individual cases.

Figure 2 gives the characteristic curve of the dark adaptation study of a normal eye. The fifteen minute reading of the right eye shows the sudden decrease in sensitivity, as noted by the increased amount of light necessary at that reading. This is not pathologic but is caused by inattention on the part of the patient. The graph shows the normal range to be between 0.000010 and 0.000100 photons. Moderate functional disturbances yield a terminal reading at thirty minutes between

42. Feldman, J. B.: A Graph for Recording Results in Dark Adaptation, Am. J. Ophth. **19**:510, 1936.

43. Jones, cited by Lythgoe.[20]

0.000100 and 0.000500. I have often found a graph similar to figure 3 in cases of vitamin A deficiency. Quite often one sees a slight loss of sensitivity during the plotting of the dark adaptation graph. To this upturn on the graph I have given the name "rod suppression."

I have often obtained readings of between 0.000500 and 0.0100000 photons in cases of severe glaucoma, choroiditis or arteriosclerosis

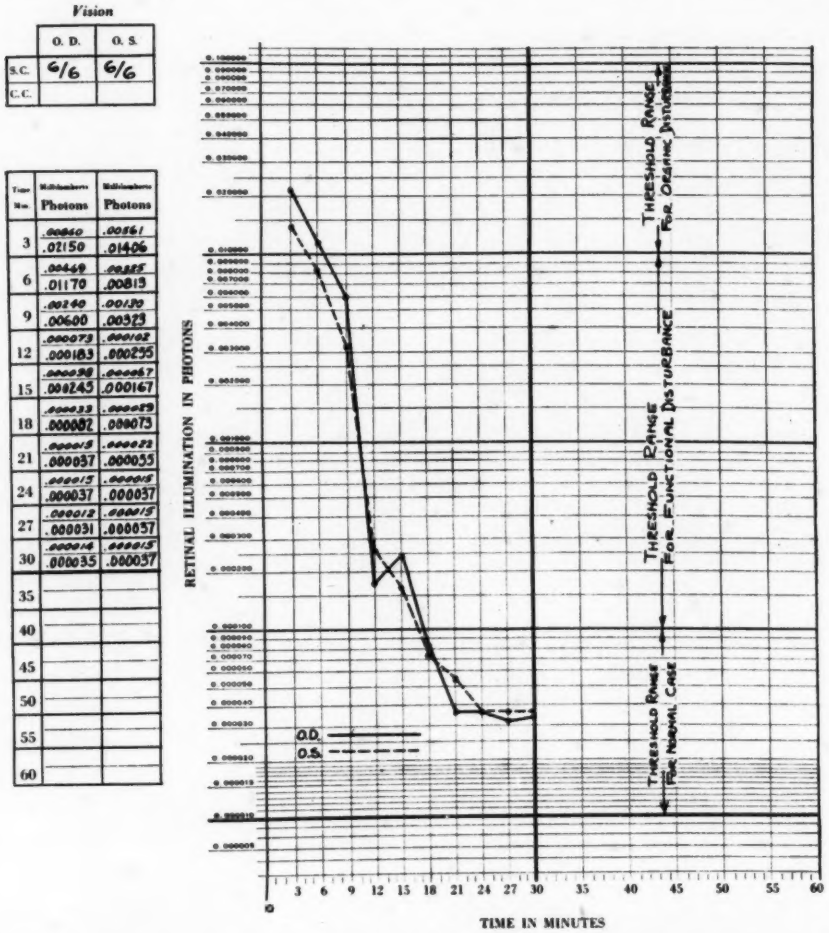

Fig. 2.—Dark adaptation graph for the normal eye, recorded in photons. The sudden rise for the right eye at fifteen minutes denotes inattention. Millilamberts and photons are recorded in the table at the left. To the right are shown the various threshold ranges of dark adaptation readings.

(fig. 4). The threshold range (i. e., the various three minute intervals of dark adaptation studies) for organic disturbances has been above 0.010000 photons (fig. 5). "Total suppression" sometimes may occur, wherein the patient is unable to see the light of the apparatus for several readings. "Total and absolute suppression" is the term used when

the patient is unable to see the light for the entire thirty minutes of study. Such readings are often found in cases of retinitis pigmentosa and choroideremia.

RESULTS

The various results which I have obtained by the quantitative machine (the latest device, the photograph of which is here shown)

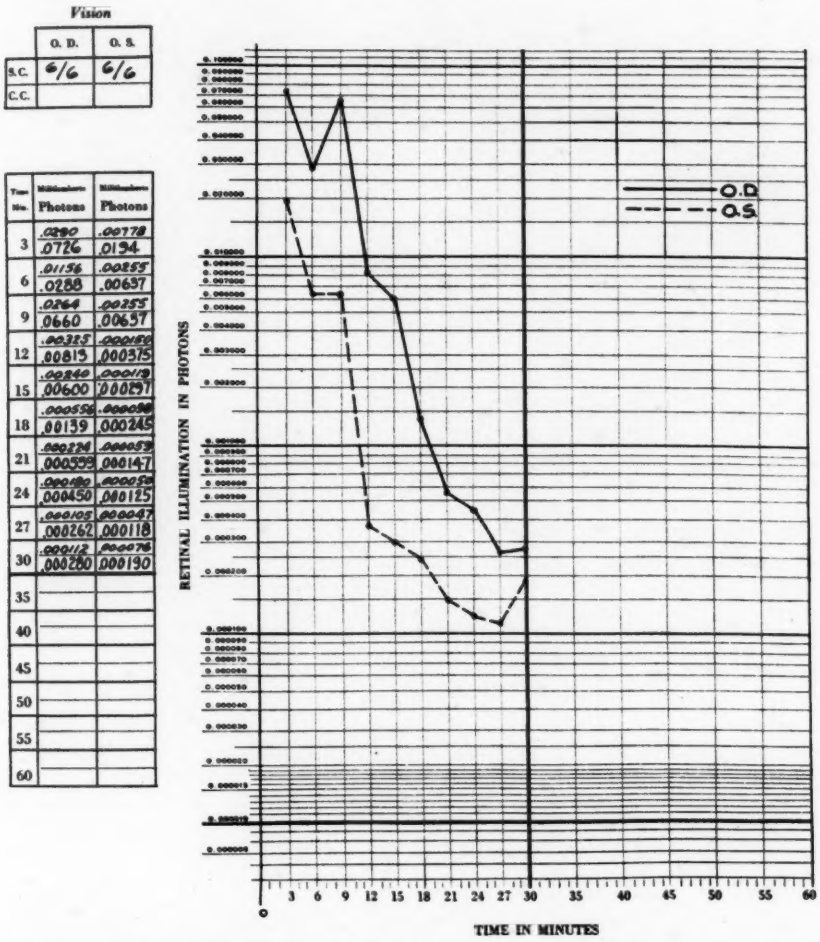

Fig. 3.—Dark adaptation graph in a case of dysaptation associated with renal calculi. Note the "rod suppression," the sudden loss of sensitivity at the thirty minute reading. This type of graph is common.

was reported in my previous papers.[44] Four thousand eyes have been examined to date. The results have been consistent and merit a continuation of my studies.

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44. Feldman, footnotes 12 and 14[a].

While there is no doubt that the tonometer and perimeter are far more useful as a diagnostic measure in cases of glaucoma, I have found two cases of early glaucoma in which the tension and the visual fields were normal, cupping was suggestive and the results of the dark adaptation study definitely showed that the eyes were not normal and

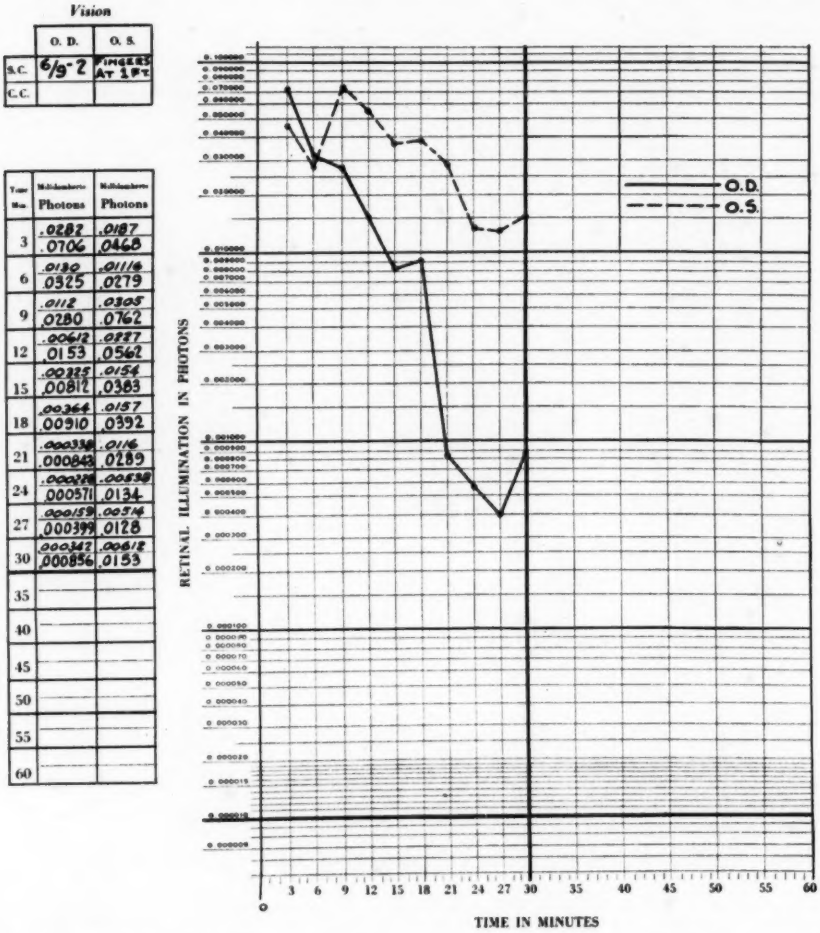

Fig. 4.—Dark adaptation graph in a case in which a provisional diagnosis was made of chronic congestive glaucoma of the left eye (tension 77) and possible simple glaucoma of the right eye (tension, 20). The two curves show the dark adaptation seen in the different stages of the disease. This study was done on the patient's admission to the hospital. After a few days the provisional diagnosis was verified.

a guarded prognosis should be given. The ensuing course in these cases verified my opinion. In both patients a frank glaucoma developed.

With the qualitative device,[25] normal dark adaptation readings were obtained for patients with alcoholism (eleven patients), arthritis of

the knee, asthma, choked disk, slight cold and hay fever, diseased tonsils, drug addiction, gonorrhea (seven patients), an infected finger, interstitial keratitis, a low intelligence quotient (ninety patients), sclerosing keratitis, syphilis (six patients) and squint (all types). Pathologic dark adaptation readings were obtained for patients with acute severe infection, arteriosclerosis, cervical adenitis, choroiditis, a high degree

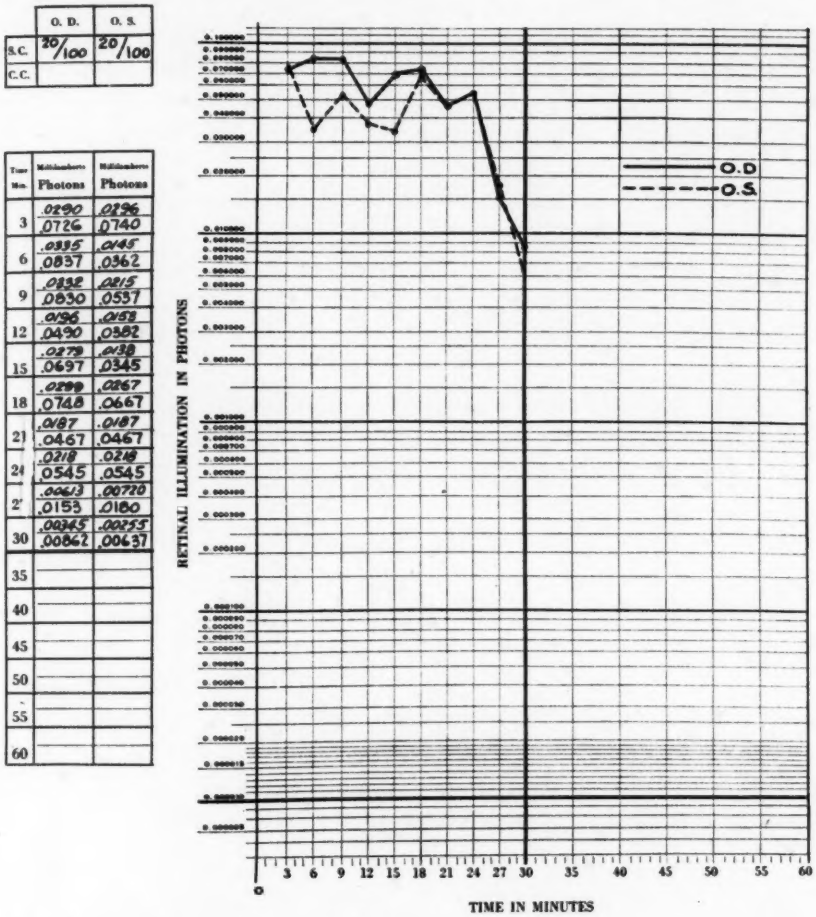

Fig. 5.—Dark adaptation graph in a typical case of retinitis pigmentosa. Sometimes the condition was so severe that the patient could not see any light for some time—"total rod suppression."

of myopia (three patients), hypertension and diabetes, hypertension and renal disease, renal calculi, sinus disease and thyroid disease. Such results were also obtained for mothers who had been nursing their children for four months, for women who were six months pregnant and for three persons who were microscopists. Six hundred and fifteen

patients were examined at three different institutions; a total of 12 per cent showed dysaptation. This study was undertaken by the ophthalmic department, St. Christopher's Hospital for Children in Philadelphia.

A few patients gave questionable results; for example, normal dark adaptation was found in one patient with choroiditis and chorioretinitis. This can be explained in this manner: Since both eyes are open during the test with this instrument, the patient saw the light stimulus with the good eye, and therefore the result was normal. A preliminary examination of the fundus, of course, aids in evaluating the patient's response. With one eye examined at a time and the light stimulus placed so as to encompass the involved choroidal area, the test would, no doubt, have shown a pathologic condition. In one of the institutions twenty patients had pathologic dark adaptation. The results of the ocular examination were negative. Under the assumption that this dysaptation was due to vitamin A deficiency, each patient was given 20,000 units of vitamin A per day. After six weeks, eighteen patients showed normal dark adaptation. Two patients showed no improvement.

SUMMARY AND COMMENT

Dark adaptation is defined.

The von Kries theory is briefly described, as is also the physiologic explanation for day and night vision and their relation to dark adaptation. Mention is made of the history of dark adaptation studies. The importance of dark adaptation is noted as a test for certain pathologic conditions of the eye, such as glaucoma and arteriosclerosis. It gives information as to the response of the rods and the visual purple to light. It is, therefore, quite useful preoperatively when an opacity of the lens makes an ophthalmoscopic examination impossible.

When persons with vitamin A deficiency or diseases believed to be the result of avitaminosis A show a diminished or total loss of regeneration of the visual purple, it is evidenced by a pathologic dark adaptation.

All patients who are to be subjected to a study of the dark adaptation are divided into groups, depending on which of the following factors is interfered with: (1) physical, (2) physiologic and (3) biochemical. The grouping is not necessarily a sharp one, and a patient may be placed in more than one group.

The fundamentals necessary for a correct instrument for recording dark adaptation are discussed, as to the range of light, size of light stimulus and, most important, the retention of the quality of the light stimulus, regardless of how low the intensity of light becomes (i.e., the wavelength of the light must not be altered). Reducing the intensity of light by altering the current in the lamp is not permissible.[22b]

The minimum light visible is best calibrated either in lambert fraction or preferably in photons, since this is primarily an ocular problem. Noiseless operation of the apparatus is an asset. The proper technic is given.

The value of ophthalmologic examination in each case in which dark adaptation is to be studied is stressed, as is also the size of the pupil in affecting the tabulation of the light sensitivity.

The various methods of controlling the size of the pupil are given.

Preexposure of the dark-adapted eye to light is recommended for the purpose of starting all examinations in a uniform manner. The advantage of preexposure to artificial light over preexposure to sunlight is given.

Attention is called to the desirability of absolute darkness in doing dark adaptation studies when certain types of instruments are used. The advisability of being able to record notations of both the time and the amount of light used in the dark room is noted.

Much time is saved by having the patient fix his eye at a certain point while the studies are being made.

A new instrument for recording dark adaptation is described.

There is a tendency to the production of rather characteristic graphs in certain diseases.

Poor dark adaptation has been noted by many observers to be of great importance in connection with the large number of automobile and aircraft accidents caused by acquired night blindness. A compulsory examination of those who drive at night might disclose any tendency to beginning night blindness, thereby proving of practical value in the prevention of the appalling accidents occurring in night travel.

707 Physicians Building.

REMOVAL OF CATARACT BY ASPIRATION

MARVIN J. BLAESS, M.D.
DETROIT

Any one moderately familiar with ophthalmic literature cannot help but be curiously impressed by the prolific number of reports regarding the efficiency of, and fine results obtained with, a multitude of different surgical technics employed for the removal of cataract. It is difficult for any one to judge impartially the best among a dozen or more different operative procedures when one is not equally familiar by adequate experience with all of them. Arruga [1] recently pointed out that an operator who possesses a practice of several years with a technic which has become his routine is not easily convinced of the advisability of changing it without reasons and facts having considerable weight. It is necessary that the technic which he does not use present advantages such that they compensate for the trouble involved in changing habits already acquired.

While the technic of cataract extraction does remain largely a matter of individual training, preference and experience among ophthalmic surgeons, it is nevertheless generally recognized that some types of cataract are more easily and more efficiently removed by some one method than by others. Clapp [2] mentioned briefly the possibility of applying a limited number of different surgical technics to the same type of cataract but inferred that the choice of method lies chiefly in the individual skill of the operator and not in the type of cataract. It seems not entirely illogical that the attainment of some measure of skill should be taken for granted in those trained to do surgical work on the eye and that more emphasis should be placed on the types of changes in the lens and the efforts to determine which particular technic is best suited to each of them. Moreover, it is presumable that the time will eventually come when a standardized general ophthalmologic training will permit one to look beyond the individuality of the surgeon and to give more consideration to the ocular changes themselves in determining the most desirable type of surgical treatment for each cataract. In

1. Arruga, H.: Les avantages et les inconvénients de l'extraction totale de la cataracte, Bull. et mém. Soc. franç. d'opht. **48**:218, 1935.

2. Clapp, C. A.: Cataract Etiology and Treatment, Philadelphia, Lea & Febiger, 1934.

a recent paper on cataract Wright [3] not only approached the matter from this standpoint but even attempted to assign to each of several types of cataract the particular surgical technic that he believed the most appropriate.

At any rate, it seems rational that entirely aside from differences in individual ability there are certain characteristics inherent in certain types or stages of pathologic ocular changes which make some surgical procedures more efficiently applicable than others.

From recent comprehensive reports,[4] from personal experience and from the studies of the results in persons operated on over a period of several years I have come to the conclusion that the factor of individualism in surgical work is somewhat overrated and that after all it is the proper application of the most efficient technic in each case considered from the point of view of the type of ocular change present that is most important. For example, a modification of the Barraquer technic [5] has been successfully adapted to the extraction of traumatic, dinitrophenol and morgagnian cataracts and to senile nuclear and senile cortical cataracts in practically all stages of incipiency and maturity. However, other technics have been employed without hesitancy for certain types of cataract when the circumstances appeared to indicate the advisability.

The particular types of cataract on which this report is based were selected on the basis of a relative lack of sclerosis of the nucleus of the lens and a certain degree of increased fluid content or softness of the pathologic lens structure, as determined chiefly with the slit lamp. In other words, an attempt was made to apply the technic only to various types of soft cataract. A lens in which the pathologic change is largely an autolysis of the lens fibers, with imbibition of fluid and little or no nuclear sclerosis, is best suited to removal by this method. However, the most important factor in selecting a suitable type of cataract for this surgical technic is the absence of a large hard nucleus; for even if the various strata of the lens are compact and contain no more than a normal amount of fluid, the technic still provides for further softening and efficient removal. The particular types of cataract encountered in this series are indicated in the table.

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3. Wright, R. E.: Lectures on Cataract, Am. J. Ophth. **20**:1 (Jan.); 119 (Feb.); 240 (March); 376 (April) 1937.

4. de Grósz, E.: L'extraction de la cataracte d'après 15,000 opérations, Arch. d'opht. **53**:161 (March) 1936.

5. Blaess, M. J.: Barbiturate Therapy and Cataract Surgery in Parkinsonism, J. Iowa M. Soc. **27**:571 (Nov.) 1937. Wolfe, O., and Blaess, M. J.: The Barraquer Intracapsular Cataract Operation, Eye, Ear, Nose & Throat Monthly **14**:200 (July) 1935.

Data on Cases of Cataract in Which Aspiration Technic Was Used

Patient	Age	Sex	Eye	Condition of Crystalline Lens	Best Corrected Vision Before Operation	Schedule of Operative Treatment	Date of Refraction and Visual Result
1	5	M	R	Congenital embryonic perinuclear cataract	Too young for refraction	8/10/34: Discission of anterior capsule 8/16/34: Aspiration 9/26/34: Discission of posterior capsule	9/25/34: 10/200* 2/ 6/35: 20/20
2	5	M	L	Congenital embryonic perinuclear cataract	Too young for refraction	10/31/34: Discission 11/14/34: Aspiration; discission of posterior capsule	2/ 6/35: 20/20
3	25	M	R	Hereditary posterior subcapsular cataract	20/200	10/12/34: Discission 10/15/34: Aspiration; discission of posterior capsule	11/ 1/34: 20/15
4	25	M	L	Hereditary posterior subcapsular cataract	20/100	10/18/34: Discission 10/23/34: Aspiration; discission of posterior capsule	11/ 1/34: 20/15
5	13	M	R	Congenital embryonic perinuclear cataract	2/200	4/11/35: Discission 4/16/35: Aspiration; discission of posterior capsule	5/17/35: 20/70 2/ 5/36: 20/20
6	13	M	L	Congenital embryonic perinuclear cataract	20/200	4/26/35: Discission 5/ 3/35: Aspiration; discission of posterior capsule	5/17/35: 20/70 2/ 5/36: 20/20
7	10	F	L	Congenital embryonic perinuclear cataract	Finger counting	4/11/35: Discission 4/18/35: Aspiration; discission of posterior capsule	5/17/35: 20/70 2/ 5/36: 20/30
8	10	F	R	Congenital embryonic perinuclear cataract	Finger counting	4/26/35: Discission 5/ 1/35: Aspiration; discission of posterior capsule	5/17/35: 20/70 2/ 5/36: 20/30
9	38	F	R	Soft cortical cataract	Hand movement	3/29/35: Discission 4/ 5/35: Aspiration; discission of posterior capsule	5/ 4/35: 20/30 5/10/35: 20/20
10	38	F	L	Soft cortical cataract	8/200	4/15/35: Discission 4/20/35: Aspiration; discission of posterior capsule	5/ 7/35: 20/30
11	25	F	L	Complicated cataract; intumescent lens and secondary glaucoma	Hand movement	4/ 1/35: Discission 4/ 5/35: Aspiration 4/11/35: Discission of posterior capsule	4/22/35: Hand movement; good central opening with fundus plainly visible; old disseminated choroiditis involving macular area

12	42	F	R	Cortical cataract	Light perception	10/ 9/34: Discission 10/15/35: Aspiration of cortex and expression of solid nucleus; discission	1/23/35: 20/20 2/21/35: 20/15
13	15	M	L	Congenital embryonic nuclear cataract	20/100	5/29/35: Discission 6/ 3/35: Aspiration; discission of posterior capsule	6/19/35: 20/30
14	40	F	L	Incipient cortical cataract	Finger counting	6/11/35: Discission 6/14/35: Aspiration; discission	7/12/35: 20/40 8/16/35: 20/20
15	13	M	R	Congenital embryonic perinuclear cataract	Hand movement	8/20/35: Discission 9/ 4/35: Aspiration and extraction of posterior capsule with forceps	10/ 1/35: 20/40 12/14/35: 20/30
16	13	M	L	Congenital embryonic perinuclear cataract	20/100	9/10/35: Discission 9/14/35: Aspiration; discission of posterior capsule	10/ 2/35: 20/50 12/14/35: 20/30
17	12	M	R	Soft cortical cataract	20/50	11/ 9/35: Discission 11/12/35: Aspiration; discission of posterior capsule	12/ 5/35: 20/50 12/19/35: 20/20
18	12	M	L	Soft cortical cataract	20/100	10/23/35: Discission 10/28/35: Aspiration; discission of posterior capsule	12/ 2/35: 20/50 12/19/35: 20/50 (amblyopia exanopsia)
19	28	F	R	Dinitrophenol cataract	Hand movement	1/23/36: Discission 1/25/36: Aspiration; discission of posterior capsule	2/13/36: 20/30 4/12/36: 20/20
20	28	F	L	Dinitrophenol cataract	Hand movement	2/ 3/36: Discission; aspiration Discission of posterior capsule	2/13/36: 20/40 4/12/36: 20/20
21	39	F	R	Soft cortical cataract	Hand movement	3/20/36: Discission 3/26/36: Aspiration; discission	4/10/36: 20/100
22	39	F	R	Dinitrophenol cataract	Hand movement	4/ 1/36: Discission 4/ 4/36: Aspiration; discission of posterior capsule	4/14/36: 20/30 4/24/36: 20/15
23	39	F	L	Dinitrophenol cataract	20/100	4/25/36: Discission 4/30/36: Aspiration; discission of posterior capsule	5/13/36: 20/15
24	25	M	R	Congenital embryonic perinuclear cataract	20/100	1/10/36: Discission 1/15/36: Aspiration; discission of posterior capsule	1/31/36: 20/20
25	25	M	L	Congenital embryonic perinuclear cataract	20/100	12/16/35: Discission; aspiration Discission of posterior capsule	1/ 8/35: 20/40 1/31/36: 20/15

Data on Cases of Cataract in Which Aspiration Technic Was Used—Continued

Patient	Age	Sex	Eye	Condition of Crystalline Lens	Best Corrected Vision Before Operation	Schedule of Operative Treatment	Date of Refraction and Visual Result
26	44	F	L	Dinitrophenol cataract	Hand movement	7/14/36: Discission 7/17/36: Aspiration; discission of posterior capsule	8/ 6/36: 20/20 10/14/36: 20/15
27	44	F	R	Dinitrophenol cataract	20/200	7/27/36: Discission 7/29/36: Aspiration; discission of posterior capsule	8/18/36: 20/50 10/14/36: 20/20
28	29	F	R	Parathyroid tetany cataract (after thyroidectomy)	Hand movement	9/24/36: Discission of anterior capsule 9/28/36: Aspiration; discission of posterior capsule	11/16/36: 20/40
29	29	F	L	Parathyroid tetany cataract (after thyroidectomy)	Finger counting	10/ 6/36: Discission; aspiration Discission of posterior capsule	Postoperative iridocyclitis 11/16/36: 20/30
30	15	M	R	Myopia gravis (no cataract)	20/100	10/ 8/36: Discission; aspiration Discission of posterior capsule	10/20/36: 20/50
31	27	F	R	Keratoconus and cortical cataract	Finger counting	8/21/35: Discission 8/26/35: Aspiration	9/ 3/35: 20/100
32	13	F	R	Congenital embryonic perinuclear cataract	Hand movement	11/28/36: Discission 12/ 4/36: Aspiration; discission of posterior capsule	12/28/36: 20/200
33	13	F	L	Congenital embryonic perinuclear cataract	20/100	12/11/36: Discission 12/16/36: Aspiration; discission of posterior capsule	12/28/36: 20/30
34	6	F	L	Left congenital circumscribed posterior capsular cataract; left microphthalmos and agenesia	Finger counting	11/27/36: Discission 12/ 5/36: Aspiration; discission of posterior capsule	12/28/36: 20/100*
35	34	F	R	Dinitrophenol cataract	Hand movement	12/11/36: Discission 12/13/36: Aspiration 1/ 8/37: Discission of posterior capsule	1/12/37: 20/20
36	34	F	L	Dinitrophenol cataract	Finger counting	12/23/36: Discission and aspiration 1/ 5/37: Discission of posterior capsule	1/12/37: 20/20
37	31	M	R	Partial traumatic cataract	Hand movement	4/22/37: Discission 4/26/37: Aspiration; discission of posterior capsule	8/26/37: 20/40 11/ 7/37: 20/20

* The patient was illiterate.

The data for all cases in which this surgical technic was attempted during a period of three years are included in the table. No case has been omitted in which the operation was performed by this particular method in the ophthalmologic service of the Evangelical Deaconess Hospital during this period. These facts are important because they enhance the value and reliability of the statistics and bear out some of my contentions. I merely wish to present a review of my actual experiences with an aspiration technic used on a carefully selected series of 37 eyes in order to obtain a critical survey of the results and to show that this technic deserves wider recognition and consideration. It is not described in any of the recent books dealing with cataract and is only briefly mentioned in one recent text.[6]

The surgical technic employed is a modification of the Teall method.[7] A number of minor but original changes have been made and some new and interesting maneuvers have been incorporated into the technic as employed in this series and described here.

It was primarily the advent of the Hildreth lamp,[8] an incident of no little importance, that brought new possibilities of a much wider range of usefulness and efficiency to the aspiration technic. The absorption of ultraviolet rays by the crystalline lens and the resulting characteristic appearance of the lens substance reveal its presence even in minute quantities and fragments. This permits the removal of the lens substance to an extent that heretofore had always been hoped for but seldom attained. Irrigation of the anterior chamber is unnecessary with this technic. It leaves nothing to guesswork and permits the surgeon to carry the operation of the removal of the softened lens material to a known and satisfactory degree of completion. Practically no dependence is placed on the faculties of absorption, the injurious and destructive effects of prolonged phacoanaphylactic inflammatory reaction thereby being avoided and the incidence of after-cataract, synechia, atrophy of the iris, and postoperative glaucoma being reduced. The technic may be carried out in one, two or three steps, depending on the individual circumstances in each case.

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6. Atkinson, D. T.: External Diseases of the Eye, Philadelphia, Lea & Febiger, 1937.

7. Wood, C. A.: A System of Ophthalmic Operations, Chicago, Cleveland Press, 1911. Dean, F. W.: Operation for Congenital and Juvenile Cataract, Tr. Am. Acad. Ophth. **31**:261, 1926.

8. The Hildreth lamp is a carbon arc lamp designed for ocular ultraviolet illumination, so that prolonged exposure of the operative field does not result in a burn, as would be the case with a more intense therapeutic lamp. I know of no other lamp made for this specific purpose. It has a quartz lens system, including a focusing lens. It was mentioned by W. A. Fisher (Senile Cataract, ed. 3, Chicago, H. G. Adair Printing Co., 1937, pp. 135-137).

The first step consists of discission of the anterior capsule and breaking up of the lens structure. The second step consists of aspiration of the softened lens substance, and the third step, of discission of the posterior capsule. A general operating lamp, which provides mixed or white light, and a Hildreth lamp, which provides ultraviolet rays, are focused on the operative field in such a way that the type of illumination is readily interchangeable, so that on instant notice either one or both may provide the illumination.

The patient is carefully prepared for operation under strictly aseptic hospital routine. It is important that the pupil be widely dilated at the beginning of the surgical treatment and kept dilated until after the last step is completed.

TECHNIC

Anesthesia is obtained by from four to six instillations of 5 per cent solution of cocaine hydrochloride at five minute intervals. With small children, an intravenous anesthetic, such as evipal sodium, may be used. Inflammable inhalation anesthetics are contraindicated in the presence of the exposed heated carbons of the Hildreth lamp. The operation is started under white light. The eyeball is fixed by seizing a fold of the conjunctiva below the cornea with Green's fixation forceps. A discission knife with a short broad blade is inserted through the cornea into the anterior chamber from the temporal limbus. Multiple broad cross lacerations involving all portions of the anterior capsule are then made by wide sweeping excursions of the knife. One must be cautious not to injure the iris or the posterior capsule at the thinner portion of the lens near the equator. After the anterior capsule has been considerably frayed, the blade of the knife is inserted more deeply into the substance of the lens, and the latter is broken up by the shorter movements of the broad portion of the knife blade held at an acute angle. This is best accomplished by short circular movements of the knife blade in successive small segments of the lens and permits aqueous to come in contact with the remotest portions of the naturally hygroscopic lens material. Care must be taken not to injure or lacerate the posterior capsule or to tear the zonular fibers, as this might disturb the vitreous and permit lens débris to become mixed with it. When the lens has been thoroughly broken up, the discission knife is withdrawn and the eye is illuminated with the ultraviolet rays, while the white light is removed. In this way one can readily determine the extent to which mechanical destruction of the lens structure has been carried out. Two per cent atropine ointment is instilled, and the eyes are bandaged. In some cases in which an intumescent cataractous lens is extremely fluid, the second step in the surgical removal is carried out immediately after discission.

Usually a variable period of time (from one to several days, depending on the fluid content of the cataractous lens) is permitted to elapse, during which the lens substance imbibes aqueous fluid which has been brought into contact with it. The broken lens material gradually becomes swollen and protrudes into the anterior chamber, where fragments of various size gather in irregular masses. At the same time there goes on a mechanical mixing or dissolving of lens protein in aqueous, so that the latter gradually assumes a clouded or milky appearance.

If nothing further were done, this process of liquefaction would be followed by natural removal of the lens material, a trifold process involving drainage through the usual channels of the anterior segment by phagocytosis and by tissue absorption.

This is a slow and complicated biologic reaction, often involving a severe phaco-anaphylactic iridocyclitis, which is destructive to the iris and eventually results in extensive atrophy of the iris, adhesions and a dense proliferative after-cataract. These are precisely the destructive and vitiating changes that the aspiration technic is specifically designed to obviate.

When the lens material has become softened, swollen and mixed with aqueous, the second step in its removal is carried out. The period of time required for adequate liquefaction, dissolution and autolysis depends to a large extent on individual circumstances. In most cases from one to several days were required to permit adequate liquefaction. In a few cases the intra-ocular pressure became moderately elevated during this period, but in no case did secondary glaucoma become a serious menace, and in a few cases in which it appeared it was entirely and permanently relieved after the aspiration of the lens débris.

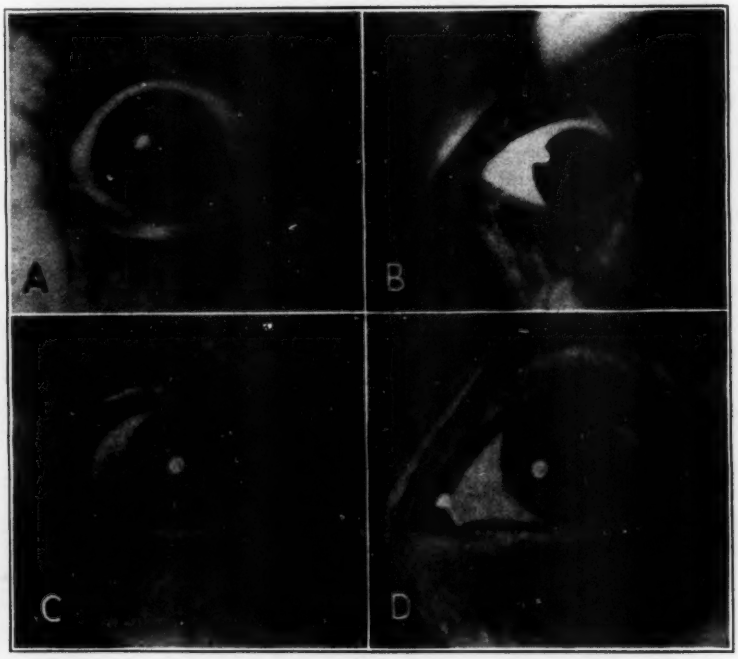
The second step in the technic is carried out under the same careful aseptic and anesthetic conditions as the first. Akinesis and retrobular injection are unnecessary but may be utilized for exceedingly unruly patients. The same type of dual illumination is used as previously described for the first step. With the lid speculum in place, the eyeball is fixed with a suitable forceps and a keratome incision, from 4 to 5 mm. in length, under a narrow conjunctival flap is made at the limbus on the temporal side. The temporal side is selected as the most convenient position where there is no anatomic structure to interfere with the insertion and manipulation of the suction end piece. The suction cannula, which is connected to a piece of rubber tubing and a mouth piece, is then introduced into the anterior chamber through the keratome incision, and by the application of oral suction the softened and partially liquefied lens débris and aqueous are aspirated into the tube. Usually there is some pupillary constriction as the lens is aspirated. Often it is necessary to maneuver the opening in the suction tip into an oblique position under the pupillary margin of the iris in order to facilitate aspiration and yet not injure the iris. Care must be taken not to tear the posterior capsule during aspiration. When the softened lens material has been removed, the suction tip is withdrawn, and the anterior chamber is permitted to refill with aqueous. When there is considerable atrophy of the iris and ciliary body, the reformation of the anterior chamber is naturally slow and may be hastened by injecting physiologic solution of sodium chloride through the keratome incision under the conjunctival flap. Care must be taken not to inject the solution under any appreciable pressure.

Next the eye is examined carefully under the ultraviolet rays. Fragments of lens débris that have escaped aspiration are immediately discovered, and their locations are noted. The posterior capsule appears as a fine whitish weblike structure under the ultraviolet radiation. Under general illumination, a second sterile suction tip is introduced into the anterior chamber, and with the aid of the ultraviolet ray lamp any remaining fragments of lens material are aspirated and the suction tip is withdrawn.

The anterior chamber is then permitted to reform again, and the third and final step in the procedure may be carried out as a continuation of the second step. A curved discission knife is introduced into the anterior chamber through the keratome incision, and a single linear vertical incision is made in the posterior capsule directly through the center of the pupillary area. This discission may be performed under the ultraviolet ray lamp, so that there need be no question as to the length and position of the opening of the capsule by the incision. A sterile syringe filled with air and fitted with a blunt-tipped lacrimal needle is now used to distend the anterior chamber with air. If the iris shows any tendency to

prolapse, it is replaced with an iris repositor. The use of air in the anterior chamber in no way interferes with healing. The air is gradually absorbed in from three to five days, during which time it holds the iris away from the cornea and prevents peripheral anterior synechiae.

Incidentally, it is surprising to many to find how well the eye tolerates air. I have used injections of from 2 to 3 cc. of air, as advocated by Arruga, directly into the vitreous cavity in several cases in which the eye became extremely soft after escape of subretinal fluid in operations for retinal detachment. When there is no retinal tear in the upper quadrants of the globe, where the air collects by gravity into a single bubble, this procedure is entirely harmless, and the air is gradually absorbed and replaced by tissue fluids. The procedure may be repeated with impunity if a subsequent operation for retinal detachment becomes necessary.



Aphakic eyes after removal of cataracts by aspiration technic. *A*, the eye in case 24; *B*, that in case 25; *C*, that in case 27, and *D*, that in case 26.

In completing the aspiration operation, usually no sutures are necessary, but if there is any gaping of the wound it is well to use a single black silk conjunctival suture. An ointment containing mercury bichloride in a concentration of 1:3,000 is instilled into the conjunctival sac, and both eyes are bandaged.

In some cases in which the patient becomes restless, in which there is hemorrhage from the wound or in which the pupil becomes markedly constricted after aspiration, the discission of the posterior capsule is not done until later, after the keratome incision has healed. In such instances the discission constitutes a separate third step in the technic.

The visual results are indicated in the table. It is interesting to note, especially in cases of congenital cataract, how vision improves with use of the eye for some time after the cataract has been removed.

SUMMARY AND CONCLUSIONS

On the practical basis of visual results, the aspiration method of cataract removal may be regarded as eminently successful. By this technic prolonged phacoanaphylactic reactions are avoided and the incidence of late postoperative complications is reduced to a minimum. It is a conservative procedure, involving only a small incision, which heals quickly, thereby limiting the dangers of hemorrhage and infection.

The technic is particularly suitable for children and for adults who are difficult to keep under quiet control. A surgeon may carry the operative work to almost any stage of completion, and if unfavorable circumstances intervene he may postpone the remaining surgical treatment until later, when circumstances become more favorable. On the other hand, the absolute quiet which is so necessary after a large corneal incision has been made is not such an important factor in the postoperative care after the use of the aspiration technic. Patients may be permitted considerable latitude in movement immediately after the aspiration operation and are permitted to be up much earlier than if some technic requiring a larger incision were used. The results obtainable by its use in carefully selected cases justify a prominent place for the aspiration technic among the conservative methods of cataract extraction.

2707 Book Tower.

SIR HANS SLOANE'S ACCOUNT OF AN EFFICACIOUS MEDICINE FOR SORENESS OF THE EYES

AN EPISODE IN EIGHTEENTH CENTURY OPHTHALMOLOGY

BURTON CHANCE, M.D.
PHILADELPHIA

Ophthalmic medicine in England in the first half of the eighteenth century was at low level. Quacks were in their heyday; Read, Grant, Taylor and other mountebanks flourished, but it was the final season of their harvesting. Surgeons like Cheselden, Sharp, Ware and others, strengthened by Daviel's success in the complete removal of cataract, began to arouse the interest of serious, honorable members of the medical profession in a scientific investigation and comprehensive understanding of the conditions affecting the eyes which they had for so long committed to the far from tender ministrations of irregular practitioners who were spoken of as "oculists" and regarded with the greatest scorn and contempt by the members of the regular medical profession. All sorts of nostrums were exploited, and society tolerated quackery from force of the example set by certain eminent physicians of the day who vended medicines the composition of which they kept secret. The great Dr. Mead possessed a secret powder (his favorite nostrum) which he sold for the bite of a mad dog.

Some time ago, while searching the index of *The Gentleman's Magazine* for 1771 for a reference on a general subject, I was astonished to see "Reflections on the Treatment of Ophthalmia."[1] Immediately abandoning my search and turning to the designated chapter, I found the article the title of which had attracted my attention. It was a rather lengthy letter from one who signed himself "Investigator," in which he criticized a method of treatment offered by that distinguished medical practitioner Sir Hans Sloane. Becoming absorbed in the contemplation of this letter, which vividly expressed a mode of therapy for active inflammation of the eye by practitioners in Western Europe two hundred years ago, I decided to make it the subject of an

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Read before the College of Physicians of Philadelphia, Section on Ophthalmology, Dec. 16, 1937.

1. Reflections on the Treatment of Ophthalmia, Gentleman's Mag. & Hist. Chron. **41**:485, 1771.

account of some of the ways diseases of the eyes were treated just before the dawn of the scientific understanding of the principles of ophthalmology.

To have found so specific a medical communication in a popular magazine was most unexpected. The magazine, however, should be of interest, the formal title being *The Gentleman's Magazine and Historical Chronical—Prodessi et Delectare—E Pluribus Unum—by Sylvanus*

THE

Gentleman's Magazine,

AND

Hiſtorical Chronicle.

VOLUME XLI.

For the YEAR M.DCCLXXI.

By SYLVANUS URBAN, *Gent.*

LONDON:

Printed at St. John's *Gate*, for D. HENRY, and ſold by F. NEWBERY, the Corner of St. *Paul's Church-Yard, Ludgate ſtreet.*

Fig. 1.—Title page of *The Gentleman's Magazine and Historical Chronicle.*

Urban Gent—London. Antedating the popular "quarterlies," *The Gentleman's Magazine*, issued monthly from 1730, published material gathered from many sources. At that time there were few medical journals published in English. Up to the opening of the nineteenth century means for the dissemination of medical knowledge were few and narrow in their scope. Authors wrote books and pamphlets, and the Philosophical Society received communications, to be published more or less fully in the transactions of the society.

After perusing the letter, I sought for Sloane's original publication and was rewarded by finding it in the library at the College of Physicians. I shall endeavor to describe it and to incorporate what "Investigator" had to offer in criticism of its provisions. It is a small pamphlet, of 17 pages; the title page is reproduced in figure 3.

The *Gentleman's Magazine:*

London Gazette
Daily Advertiser
PublicAdvertiser
Public Ledger
Gazetteer
St James's Chron
London Chron.
General Evening
Whitehall Even.
London Evening
Lloyd's Evening,
Monday, Wednesday, Friday.
Oxford
Cambridge
Reading
Northampton
Birmingham 2
Bath 2 papers
Coventry 2
Briſtol 3

St. JOHN's Gate.

York 2 paper
Dublin 2
Newcaſtle 2
Leeds 2
Edinburgh
Aberdeen
Glaſgow
Ipſwich
Norwich
Exeter
Glouceſter
Saliſbury
Liverpool
Sherborn
Worceſter
Stamford
Nottingham
Cheſter
Manchester
Canterbury
Chelmſford

For NOVEMBER, 1771.

CONTAINING,

More in Quantity and greater Variety than any Book of the Kind and Price.

Foreign Affairs.—Particulars of the ſeizure and deliverance of the King of Poland 478
—Outrages at Moſcow, on account of an epidemical diſtemper there ib.
Debates in a Newly eſtabliſhed Society 479
—L. B—n's motion for augmenting the army ib.
—Gen. C—y's motion for repairing the Garriſons, &c. ib.
—Mr. O—w's motion for clearing the H. 481
—Mr. D—g's motion for examining the Journals of the Houſe of Lords ib.
—Mr. S—r's motion that the Committee might not proceed on the Land-tax for 1771 483
K. Charles I. not the Author of the Eikon Basilike 484
Reflections on the treatment of Ophthalmia 485
An Entomologiſt's anſwer to Eubius 486
Addreſs to the Commiſſioners of Yarmouth Haven 487
Tranſlation of Mr. Hoel's Journey to Stockholm 488
Second Vindicat of Mr Emerſon's Athron. 490
Crit. Remarks on ſome paſſages of Voltaire 493
Account of the Lithargus of Avignon 495
Curious Antiquities in Scotland deſcribed 496
Addreſs to the Author of Remarks on Volt. 496
Dr. Percival on ulcerated Sore Throats 497
New Verſions of the Lſt's. Pr. with Rem. 499
On the progreſs of Vegetation in Trees 500
Artifice of the Thing explained ib.
On Dr. Hewſon's enquiry into the properties of the blood 501
On the miſtakes of eminent Authors 503
Queſt. relative to the formation of mould 504
Meteorological Diary of the Weather 505
REVIEW OF BOOKS.—The Complete Engliſh Farmer ib.
—A Journal of a Voyage Round the World in his Majeſty's Ship the Endeavour 509
—Hiſtory and Memoirs of the Society formed at Amſterdam in favour of perſons ſuppoſed to be drowned 512
Catalogue of New Publications 514
POETICAL ESSAYS.—Verſes left in a Coffee houſe at Cambridge —Ode to Sleep —Alcino —On the death of Miſs Burton 515
—The Roſe Wife—Tranſlation of Hor. Ode xiii.—A ſentimental Thought 516
HISTORICAL CHRONICLE.—Liſt of Births, Marriages, Deaths, Preferments, &c.

Embelliſhed with a Plan of the River SALWARE, and of the NAVIGABLE CANAL, from Droit wich, to the River Severn, in Worceſterſhire.

By *SYLVANUS URBAN*, Gent.

LONDON, Printed for D. HENRY, at St. JOHN's GATE.

Fig. 2.—Table of contents of the issue of *The Gentleman's Magazine and Historical Chronicle* in which "Reflections on the Treatment of Ophthalmia" appeared.

Although I had known of Sir Hans Sloane's exalted position in medical and philosophic life in Europe in his century, until I saw "Investigator's" serious criticism I was not aware that he had been interested in any phase of ophthalmology. How came he in a position

to publish with authority a work in ophthalmology for which he has been given flattering notices in works of reference? In 1745 Sir Hans Sloane, physician to King George II, did publish "An Account of a Most Efficacious Medicine for Soreness, Weakness and Several other Distempers of the Eyes," basing the account on "the surprisingly beneficial effect of the use in my hands on a large number of patients" of a remedy which he ordered should be made in the following manner:

AN

ACCOUNT

OF A MOST

Efficacious Medicine

FOR

SORENESS, WEAKNESS,

And Several Other

DISTEMPERS of the EYES.

BY

Sir *HANS SLOANE*, Bart.
Phyſician to his Majeſty, &c.

LONDON:

Printed for Dan. Browne, at the *Black-Swan*, without *Temple-Bar*. Mdccxlv.

Fig. 3.—Title page of Sir Hans Sloane's book.

Take of prepared Tutty, one Ounce; of *Lapis Hæmatites* prepared, two Scruples; of the best Aloes prepared, twelve Grains; of prepared Pearl, four Grains. Put them into a Porphyry, or Marble Mortar, and rub them with a Pestle of the same Stone very carefully, with a sufficient Quantity of Viper's Grease, or Fat, to make *A Liniment;* to be used daily, Morning or Evening, or both, according to the Conveniency of the Patient. . . .

This account of his most "Efficient Medicine for the Cure of Sore and *Weak Eyes*" was made public for the "Benefit of Mankind; and

most humbly Dedicated to his MAJESTY." In the introduction, Sloane stated:

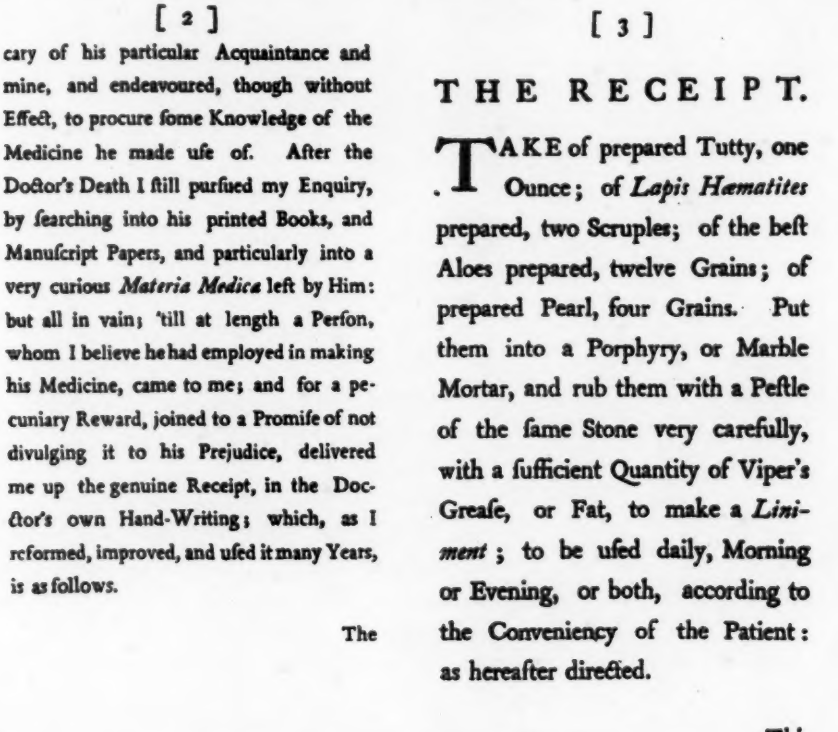
> . . . having been led by a strong natural Inclination to the Practice of Physic, through an earnest Desire to be useful in my Profession, I was always very attentive to Matters of Fact, and the real Cures that fell under my Observation. Of these, I saw many performed upon *Sore Eyes* by Doctor *Luke* Rugeley; whereupon I applied to a very understanding Apothecary of his particular Acquaintance and mine, and endeavoured, though without Effect, to procure some Knowledge of the Medicine he made use of. After the Doctor's Death, I still pursued my Enquiry, by searching into his printed Books, and Manuscript Papers, and particularly into a very curious *Materia Medica* left by Him; but all in vain; 'till at length a Person, whom I believe he had employed in making his Medicine, came to me; and for a pecuniary Reward, joined to a Promise of not divulging it to his Prejudice, delivered me up the genuine Receipt, in the Doctor's own Hand-Writing; which, as I reformed, improved, and used it many Years, is as follows [composed of the ingredients already quoted].
>
> This Medicine I soon tried, and, though a Composition, found it so surprizingly beneficial, that by the right Use of it not one in five Hundred missed of a Cure; unless their Disorder proceeded from a Venereal Taint.

"Investigator" was skeptical of Sloane's statement of his results and declared it to be "most certainly necessary to examine supposed facts before we admit their validity." The object of his letter was "to candidly enquire into the effects of the remedy recommended for an Inflammation of the eye, under the sanction of that great man." "Investigator" believed that "in few cases can the wrong application of topical remedies be productive of more lamentable prejudice than to the organ of vision," for this reason, he concluded, "I therefore should be exculpated." And he stated further, because "humanity was Sloane's motive for publishing it, humanity is my motive solely for considering it at that time when numbers were laboring under the complaints for which this medicine was said by Dr. Sloane himself, to be 'so surprisingly beneficial.'"

"Investigator's" experience with this liniment was disappointing. "In general," he stated, "when I have tried it, so much injury has been sustained, that I have been thereby lead to examine more particularly, the several ingredients of which it is composed." He learned that "tutty contains an argillaceous earth, probably with some proportion of the semi-metal called zinc, and a small quantity of lead. The last appears to have a sedative effect and in that view may be admissible; how far the zinc and the argillaceous basis of Tutty might prove hurtful cannot be wholly ascertained. *Lapis Hæmatites* is a rich iron ore, and must without doubt, prove highly stimulating, increase the inflammation, and thereby produce the most alarming effects. *Aloes* internally is stimulating, and by its effects when applied to ulcers, it is probable that it possesses the same quality when used topically in Inflammation of the

Eye. Pearl, Prepared, is ordered in so small a proportion, that little or no effect can be expected from it; it may, perhaps, act as a mechanical stimulus to the tender coats of the eye. If the *Viper's grease* be not rancid, like oil, it may relax, and thereby prove useful; but most of the other ingredients are evidently calculated to increase inflammation and pain and are therefore inadmissible."

I too, sought, to learn of what the remedy was composed. In modern terms, "tutty" is crude zinc oxide, a brownish, powdery substance obtained from the flues of smelting furnaces. It was known in the Middle Ages and was used by the Arabians, as references can be found in the earliest Arabian works on ophthalmology.



[2]

cary of his particular Acquaintance and mine, and endeavoured, though without Effect, to procure ſome Knowledge of the Medicine he made uſe of. After the Doctor's Death I ſtill purſued my Enquiry, by ſearching into his printed Books, and Manuſcript Papers, and particularly into a very curious *Materia Medica* left by Him: but all in vain; 'till at length a Perſon, whom I believe he had employed in making his Medicine, came to me; and for a pecuniary Reward, joined to a Promiſe of not divulging it to his Prejudice, delivered me up the genuine Receipt, in the Doctor's own Hand-Writing; which, as I reformed, improved, and uſed it many Years, is as follows.

The

[3]

THE RECEIPT.

TAKE of prepared Tutty, one Ounce; of *Lapis Hæmatites* prepared, two Scruples; of the beſt Aloes prepared, twelve Grains; of prepared Pearl, four Grains. Put them into a Porphyry, or Marble Mortar, and rub them with a Peſtle of the ſame Stone very carefully, with a ſufficient Quantity of Viper's Greaſe, or Fat, to make a *Liniment*; to be uſed daily, Morning or Evening, or both, according to the Conveniency of the Patient: as hereafter directed.

This

Fig. 4.—Introductory pages of Sir Hans Sloane's book.

Alas for Sloane's vaunted primacy! "Several Years after I had been in Possession of this Secret," he tells us, "in turning over some Manuscripts of Sir *Theodore Mayerne,* I found Mayerne had known the same Ointment, or Liniment, and had entered it in his *Pharmacopœa,* under his own Name: though I afterwards discovered, that it was not originally his, but had been communicated to him by Sir *Matthew Lister,* a Member of the College of Physicians; who had performed a Cure with it on a Lady *Savile,* which Sir *Theodore* thought a very extraor-

dinary one. And 'tis very probable, that he afterward communicated it to Dr. *Thomas Rugeley;* as I found they were Contemporaries, and Friends.

"The Method, which best succeeded with me in facilitating the efficacious Use of this Liniment, is to bleed, and blister in the Neck and behind the Ears, in order to draw off the Humors from the Eyes; and afterwards, according to the Degree of the Inflammation, or Acrimony of the Juices, to make a Drain by Issues between the Shoulders, or a perpetual Blister."

"Investigator" maintained "unless we make blisters perpetual, an Ophthalmia of long standing is seldom much relieved by them. The first or serous discharge of a blister, and the matured pus of an issue appear to have different effects. The latter I have found much more beneficial, but in the manner Sir Hans Sloane directs, between the shoulders, it is extremely troublesome; and I have therefore tried it behind the ear, just below the *processus mammillaris.* We have there a fleshy part as suitable as if designed by nature for such a purpose. It is about that part that nature forms frequently a drain in young children, the utility of which is known to every practitioner in medicine.

"The easiest manner of making an issue here, is by means of a caustic, the *causticum commune fortius* of the London Dispensatory. When the eschar produced by it is digested out, a pea, or half of one, may be introduced, and the part dressed in the usual manner." For washing the eyes, Sloane "generally recommended Spring Water; which he thought preferable to any spirituous Lotion, whether simple or compound."

"Investigator" hesitated to enumerate remedies for external application to the eye. He had known pain to be mitigated by a lotion made of the decoction of poppies. Spring water, solutions of vitriol, alum, sugar of lead and the vegetable solution of M. Gouillard had likewise relieved pain; he had also used poultices of bread, roasted apple, curd of milk, conserve of roses and various preparations of *lapis calaminaris,* and "tutty." But for an obstinate ophthalmia, the best of them proved ineffectual. "And the best inward Medicines," Sloane stated, "I have experienced to be Conserve of Rosemary Flowers; Antiepileptic Powders, such as *Pulvis ad Guttetam;* Betony, Sage, Rosemary, Eyebright, Wild Valerian Root, Castor, washed down with a Tea made of some of the same Ingredients; as also Drops of *Spirit · Lavendulae composit · and Sal volat · oleos.*

"If the Inflammation returns, drawing about six Ounces of Blood from the Temples by Leeches, or Cupping on the Shoulders, is very proper. The Liniment is to be applied with a small Hair Pencil, the Eye winking or a little opened."

"Investigator," commenting on these directions, pointed out that Dr. Sloane resorted to other well known effectual remedies in an ophthalmia. He himself was cautious in applying leeches to the temples, as he had found it difficult to stop the blood and to prevent the cold so liable to develop in the patient from the application of wet cloths to take up the blood, and that this method was not convenient and frequently increased the inflammation. He therefore substituted cupping, without any of the inconvenience, which could be repeated as occasion required.

[6]

Caſtor, *&c.* waſhed down with a Tea made of ſome of the ſame Ingredients: as alſo Drops of *Spirit. Lavendulæ compoſit.* and *Sal volat. oleos.*

If the Inflammation returns, drawing about ſix Ounces of Blood from the Temples by Leaches, or Cupping on the Shoulders, is very proper.

The Liniment is to be applied with a ſmall Hair Pencil, the Eye winking or a little opened.

In proſecuting the Cure of ſore Eyes, I have been ſometimes ſurprized by want of Succeſs; 'till at length I found, that the Cauſe was a lurking intermitting Fever, every Fit of which affected the Eyes, and rendered their Diſorder obſtinate: wherefore upon taking off the Fever by a proper Uſe

[7]

Uſe of the Bark, the Cure has been effectually performed.

This Medicine has cured many, whoſe Eyes were covered with opake *Films*, and *Cicatrices* left by Inflammations and Apoſtems of the *Cornea*; which, though they happen to Perſons of all Conditions, yet are more common among the poorer Sort of People: many of whom were ſo totally deprived of Sight, as to be under a Neceſſity of being led to me; and after ſome time could perfectly well find their Way without a Guide, to my great Satisfaction. And it is not only very beneficial in ſuch Caſes, but alſo where there is an exceſſive Pain in the Eyes, ſhooting thence up into the Head: as I particularly remember in a great Lady, who had ſuch Pains in her ſore Eyes, and Head, that ſhe had, when I firſt ſaw her,

Fig. 5.—Pages on therapy in Sir Hans Sloane's book.

Sloane was "sometimes surprized by want of Success; 'till at length I found, that the Cause was a lurking intermittent Fever, every Fit of which affected the Eyes, and rendered their Disorder obstinate: wherefore upon taking off the Fever by a proper Use of the Bark, the Cure was effectually performed.

"This Medicine has cured many, whose Eyes were covered with opake *Films,* and *Cicatrices* left by Inflammations and Apostems of the *Cornea;*—common among the poorer sort of People: many of whom were so totally deprived of Sight, as to be under a Necessity of being led to me; and after some time could perfectly well find their Way without a Guide, to my great Satisfaction."

Alas! In such cases as these "Investigator" noted that "the mechanical attrition of the ingredients against such films may be useful, but these complaints are widely different from Ophthalmia. I have frequently known powdered glass blown into the eye, of the greatest benefit in such diseases, but no prudent man would recommend the same remedy in a painful inflammation of that organ."

"It is not only very beneficial in such Cases," Sloane continued, "but also where there is an excessive Pain in the Eyes, shooting thence up into the Head: as I particularly remember in a great Lady, who had such pains in her sore Eyes, and Head, that she had, when I first saw her, taken about fifty Drops of *Laudanum* thrice in twenty-four Hours, of which Complaints she and many others had been relieved by this Medicine, without the help of any Opiate."

In "Investigator's" practice, "when the pain accompanying an Ophthalmia comes on periodically, with some degree of fever, bark is given internally with great advantage."

Sloane cited the case of a commissioner, who brought recommendatory letters from friends in France, in whose eyes he observed "a great Soreness and Weakness, of which the Application of my Medicine very soon cured him. Whereupon Monsieur *Anisson* assured me, that he would procure me from the King his Master any Reward I should think fit to ask for the Secret; the King being accustomed to oblige his Subjects that way." "But," stated Sloan, "I answered him that I was then bound by a Promise to conceal it."

Incidentally, Sloane observed, "Cathartics, especially with the Addition of Mercury, are prejudicial in the Diseases of the Eyes, which are cured by this Medicine."

But "Investigator" stated, "In obstinate inflammations, I have sometimes seen mercurials of service, particularly Van Sweeten's sublimate solution. No hesitation can be made to giving this medicine very early in this complaint, especially where either any scrophulous or venereal taint is suspected."

Sloane continued, "It is also worthy of Remark, that People afflicted with weak Eyes are over-fond of Hoodwinking, or covering them from the Light; which sometimes retards the Cure, by keeping their Eyes too warm: and therefore I have constantly advised them to throw away these Coverings, as soon as they could possibly bear the Light." "Investigator," on the contrary, usually found it necessary to adjust "a slight piece of silk to prevent the too powerful rays of light, and the cold air, from affecting the inflamed eye."

Sloane's knowledge of the "great Virtue of *Viper's Grease or Fat*, in the Cure of Diseases of the Eyes" was acquired from several sources. He was intimately acquainted with *"William Stokeham*, a very ingenious

Gentleman Physician to King *William III* who had formerly spent some years at *Padua,* acquainted with the most eminent Physicians of that University," the first to tell him of it; it is recommended for that Purpose by *Daniel Ludovicus,* in his Book *De pharmacia moderno seculo applicando,* Gothae, 1671, 12°."

In the original receipt, hog's lard was named, but Sloane substituted "the Grease or Fat and found, that it added so much to the Efficacy of the Medicine, as to make it do, what I thought, Wonders, And as I was not bound to Secrecy, with regard to this Improvement, I communicated it to the late Dr. *Arbuthnot:* who, after repeated Trials of that Fat alone on sore Eyes, had so high an Opinion of its Virtues, that he looked on it as equally beneficial with the whole Medicine."

Sloane had "read in some *Missionary Letters,* that Serpent's Fat is used by the *East Indians,* for the same Purpose." He stated, "It is an Observation made by many Naturalists, that those Serpents cast their skins every Year, and with them the Coverings of their Eyes: but how far, or whether at all, their Fat is concerned in this *Phenomenon,* I leave to others to determine."

To this experience of Dr. Arbuthnot "Investigator" heartily subscribed, and he believed that "the addition of the other ingredients to Viper's fat only rendered the liniment stimulating and dangerous in a real Ophthalmia."

Sir Hans continued, "One of the most eminent and learned Chirurgical Authors, to whom I had a particular Regard in my Practice, cautions his Readers against the Use of Oil in Diseases of the Eyes; by which, I suppose he meant Olive-Oil." Here follows a most naive remark: "Agreeable to this Caution, I confess, I have never used any Oil, either alone, or mixed with other Medicines, for the Eyes; being unwilling to try Remedies, whose Effects were doubtful, and may possibly be pernicious: which I afterwards observed to be the Case with regard to Olive-Oil, when used by some People (to whom I had given the Liniment) in order to make it more liquid, upon drying; for the Liniment, in that State, has caused very considerable Complaints. This, in my Opinion, proceeds from some hot, sharp Particles, contained in the Oil; though it is generally reckoned very soft and mild in outward and inward Use: for I remember, when at *Montpellier,* I was informed by Monsieur *Magnol,* and others of my Acquaintance there, that the feeding of tame Rabbits with Olive Leaves, in want of other Food, has caused their making bloody Urine."

Sloane added, "I had formerly, as already said, promised Secrecy with regard to this Medicine: which I have religiously kept 'till now, that I think myself, for many Reasons, abundantly absolved. I have had some other Medicines of the like Nature communicated to me under the same Restriction: and exclusive of these few Cases, I cannot charge

myself with making the least Mystery of my Practice. For in Consultations, in a Number of Cases of Importance, I have been always very free, and open; far from following the Example of some Physicians of good Morals and great Reputation, who have on many Occasions thought proper to conceal Part of their own acquired Knowledge alledging the Maxim: *Artis est celare Artem.* And that I have not been inclined to conceal or monopolize Medicines of great Use, the following Instance will sufficiently show."

Sloane ends this small book by recounting the tale of a secret medicine in the possession of a certain family with which "they infallibly cured all Men and Beasts, bit by Mad Dogs and other Creatures." As this was a "Matter of Great Consequence to the Public," Sloane sought for an account of it, which he states he published in the *Philosophical Transactions* No 237. It seems that the receipt called for an ingredient well known to be a "pernicious vegetable," which was mistaken "for some other harmless plant, and notified botanists of the error and recommended to them to provide a Quantity of the *Lichen,* sufficient to answer all Occasions." He added, "It was accordingly made use of with the same Success, with which it has ever since been attended."

This pamphlet was the only separate medical work published by Sloane. A second edition was published in London in 1750 and one in French, "Histoire d'un reméde très efficace pour la foiblesse et rougeur des yeux," in Paris in 1746.

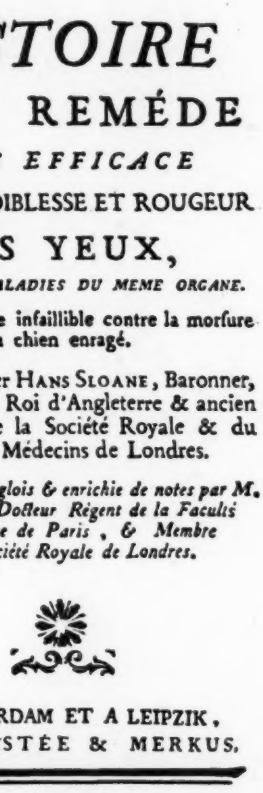
The College of Physicians possesses another edition in French, by M. Cantwell, a member of the Royal Society of London. It was published in Amsterdam by Arkstee and Merkus in 1767 and contains copious notes describing and endorsing Sloane's therapeusis. This edition is bound with a serious work by Saint Yves.

It is extraordinary that this pamphlet was for so many years held in great estimation and was republished. There is not a line concerning pathologic descriptions or the slightest inkling as to the symptoms which Sloane diagnosed as "ophthalmia."

That so famous a man as Sir Hans stooped to purchase a secret remedy, the ingredients of which he never sought to analyze, for use in as delicate an organ as the human eye is a sad commentary on the state of practice. That he, the companion of the highest-placed scientists of his day and himself capable of serious study and research in certain of the physical sciences and natural history, should have had so little regard for the conservation of his fellow men as to prescribe his "tutty compound" for hundreds of afflicted persons is one of the sad notes in that benighted era.

By this time one may rightly ask, "And who was the author of this singular treatise?'" Every one knows that Sir Hans Sloane was one

of the notable physicians of the seventeenth and eighteenth centuries, but he has not been generally classed with those who became distinguished as ophthalmologists. The only separately published medical work which I have yet been able to find is this one on the eyes, a truly singular document which might be accepted as one of the landmarks in the history of English ophthalmology. This work must have been accepted by practitioners on the authority of its distinguished author, whose position in the scientific and social circles in England was



HISTOIRE
D'UN REMÉDE
TRE'S EFFICACE
POUR LA FOIBLESSE ET ROUGEUR
DES YEUX,
ET AUTRES MALADIES DU MEME ORGANE.
Avec un Reméde infaillible contre la morsure du chien enragé.
Par le Chevalier HANS SLOANE, Baronner, Médecin du Roi d'Angleterre & ancien Préfident de la Société Royale & du Collége des Médecins de Londres.
Traduits de l'Anglois & enrichie de notes par M. CANTWEL, Docteur Régent de la Faculté de Médecine de Paris, & Membre de la Société Royale de Londres.
A AMSTERDAM ET A LEIPZIK,
Chez ARKSTÉE & MERKUS.
M. DCC. LXVII

Fig. 6.—Title page of the French edition of the book by Sir Hans Sloane.

unquestioned for over fifty years. The first remonstrance I have found was that of "Investigator" in *The Gentleman's Magazine*.

Sloane was born in Ireland in 1660, of an English mother and a Scotch father. The precocious lad early devoted himself to natural history, until, when about 16 years of age, he was seized with hemoptysis. Recurrent attacks until his ninetieth year caused him to cease all studies for three years. In 1679, arriving in London, already a well read student, he seriously resumed his researches in botany and the physical

sciences, immediately attracting to himself the foremost men of his day, who encouraged and advanced him in his favorite studies. In a year or so he went to France to pursue his investigations, and while there was induced to enter the practice of medicine. On his return to England he became associated with Sydenham, in whose house he resided for a time and through whose support he soon obtained a footing in London as a physician.

Fig. 7.—Sir Hans Sloane.

In 1685, already a fellow of the Royal Society, he sailed to Jamaica in the capacity of personal physician to the newly appointed governor of that island. The voyage from England offered much to the young man, for he visited all the islands in the Caribbean Sea. The governor died a few months after reaching Jamaica, but Sloane remained on the island to collect plants and study the indigenous animals. He applied himself assiduously to his collecting and on the return voyage classified his 800 specimens and outlined a "Natural History of Jamaica," which

he afterward published, the first volume appearing in 1707 and the second in 1725.

Appointments and honors came fast. He became associated with nearly all the hospitals, serving them faithfully and contributing to their funds. He was president of the College of Physicians; secretary of the Royal Society, and, on the death of Sir Isaac Newton, president of the society. King George II created him baronet, the first physician to be so knighted, and, besides serving as physician general of the army, in 1727 he was appointed physician to the king.

His constitution was always delicate, yet he survived all attacks of disease and lived to the great age of 92, dying at Chelsea in January 1753. All his life he had been a collector. His house in Bloomsbury became a crowded museum. When about 50 years of age he purchased the Apothecaries' Gardens at Chelsea, where he later removed his collections and took up his residence. He deeded the gardens to the Apothecaries' Society, with the understanding that they should be maintained. And so they are to this day, devoted to the promotion of botanic knowledge and especially to the cultivation of curious and rare plants.

At his death his museum and library of 50,000 volumes and 3,500 manuscripts were purchased by the nation for 20,000 pounds. This was the commencement of the British Museum.

He contributed numerous reports to the *Philosophical Transactions,* but his greatest work was the "Natural History of Jamaica."

Sloane should be honored for advocating and using the Jesuits' cinchona bark and for giving inoculations in his efforts to combat smallpox.

The wayfarer in London is reminded of him, as his name has been perpetuated in streets, squares and crescents and even by a telephone exchange!

ENCEPHALITIC OPTIC NEURITIS AND ATROPHY DUE TO MUMPS

REPORT OF A CASE

CHARLES M. SWAB, M.D.
OMAHA, NEB.

As the field of practice in a specialty is naturally more or less restricted, there are a variety of aspects of certain diseases and their complications which rarely come under observation. This statement of fact is particularly true of the specialties that have to do with the consequences of mumps. By way of introduction, a brief summary of the general subject may serve the twofold purpose of furnishing a background for the case to be reported and presenting data that are not regularly observed and noted in routine work. To delve even cursorily into the published material on epidemic parotitis brings conclusive evidence that little of consequence has been added to the general subject by present day observers, as the field has been thoroughly investigated and the various findings recorded. It is, in itself, not a new topic, as the disease was well known even in the time of Hippocrates. He alluded to the absence of suppuration in the parotid gland and also to the fact that orchitis sometimes followed parotitis. In all ages outbreaks of mumps have been both numerous and extensive, especially among soldiers in garrisons and among youths in institutions like the old-fashioned boarding schools. The usual age of incidence, however, is that of childhood, as children are gregarious and, owing to the nature of school contacts, the contagion is more likely to spread. That age does not insure immunity is evident from the fact that persons in the ninth decade have been afflicted.

Of chief interest here, however, is a consideration of the complications of mumps. Orchitis is so common and simple in its manifestations as to require little comment; oophoritis, however, though less frequent, may prove to be a far greater hazard to the patient. I recall a case in which oophoritis developed in a 4½ year old girl after mumps; peritonitis resulted and eventually death. Involvement of the central nervous system is not uncommon, but in such instances the chances of ultimate complete recovery are favorable. By way of exception to this general statement, I note and even stress the inflammations that attack the auditory and optic nerves.

Read at a joint meeting of the Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society and the Kansas City Society of Ophthalmology and Oto-Laryngology, Nov. 18, 1937, Omaha.

When disease of the central nervous system complicates the primary parotitis, either the meninges or the brain tissue may be the chief site of the inflammation; in some cases there is evidence of both meningitis and encephalitis. From a review of about 250 cases of parotitis with involvement of the nervous system, McKaig and Woltman [1] found encephalitis to be of rather frequent occurrence. Quadriplegia may be associated with paralysis of the cranial nerves, as in the case of a 7 year old child reported by Revilliod; [2] there were paralysis of both abducens nerves and of the left facial and the right hypoglossal nerve, in addition to paralysis of the extremities. The sixth cranial nerve is the most susceptible of those controlling ocular motility. With respect to the auditory nerve, Hubbard [3] expressed the opinion that from 3 to 5 per cent of the deaf-mutism in the United States is due to mumps. The sudden onset of deafness is believed by some to follow an exudate occurring in the labyrinth. Pathologic examination of the inner ear has been recorded by Toynbee,[4] who found complete disorganization of the neural apparatus in the labyrinth and a dark fluid in the vestibule and cochlea.

Many writers have presented in detail their observations in regard to the ocular complications of parotitis. Woodward [5] published an extensive account of these data and drew attention to cases of dacryoadenitis, iritis, retrobulbar optic neuritis, night blindness, conjunctivitis, acute dacryocystitis and keratitis. Butler [6] reported 2 cases of paralysis of accommodation with paralytic mydriasis, but added that there was complete recovery in both. Larkin [7] gave a detailed report of a case of bilateral choked disk. Woodward [5] also described the histologic changes in an eye that had been blinded by parotitic optic neuroretinitis three and one-half years before it was enucleated for secondary glaucoma and staphyloma. Section of the optic nerve showed complete atrophy, replacement with hyalin and obliteration of the small vessels of the nerve sheath.

REPORT OF CASE

On Jan. 18, 1937, I was consulted by M. J. L., a white man aged 25, referred to me by Dr. L. D. McGuire. The complaint at the time was loss of vision in

1. McKaig, C. B., and Woltman, H. W.: Neurologic Complications of Epidemic Parotitis: Report of a Case of Parotitic Myelitis, Arch. Neurol. & Psychiat. **31**:794 (April) 1934.

2. Revilliod, L.: Rev. méd. de la Suisse Rom. **16**:756, 1896.

3. Hubbard, T.: Tr. Am. Otol. Soc. **13**:451, 1915.

4. Toynbee, cited by Mauthner, O.: Arch. f. Ohren-, Nasen- u. Kehlkopfh. **87**:223, 1912.

5. Woodward, J. H.: Ann. Ophth. **16**:7, 1907.

6. Butler, T. H.: Brit. M. J. **1**:1095, 1930.

7. Larkin, W. R.: Illinois M. J. **38**:133, 1920.

each eye. Inquiry brought out the following history: On Nov. 28, 1936, while working in the south, the patient contracted bilateral parotitis. After he had remained in bed for several days, with the temperature reaching a maximum of from 102 to 103 F. almost daily, the right testicle became swollen and painful. The period of convalescence was uneventful until Jan. 4, 1937; on arising that morning the patient discovered that he could see only a blur of light with his right eye. Nine days later a similar condition occurred in the left eye. There was a deep-seated soreness behind the eyeballs; this had come on with the loss of vision, and persisted. Tonsillectomy and adenoidectomy were performed by a physician in Mississippi on January 6.

When the patient was first observed by me his visual acuity was reduced to faulty projection of light. The external ocular structures were normal except for widely dilated pupils, which were inactive to the usual stimuli. Study of the fundus of the right eye showed a swelling of the nerve head of 3.5 diopters. Although the retinal veins were markedly engorged, there were no exudates or hemorrhages in the retina. Examination of the fundus of the left eye showed similar changes, but with 1 diopter less of swelling of the optic disk.

Additional data were obtained by Dr. McGuire and Dr. A. E. Bennett, both at examinations in their office and during the period in which the patient was in the hospital, from January 20 to February 2. A general neurologic study revealed hyperactive tendon reflexes throughout. The first spinal puncture showed a range of pressure of from 10 to 12 mm. of mercury; subsequent punctures indicated a gradual lessening of pressure, although manometric readings were not made. There were 5 cells per cubic millimeter, and the protein content was twice normal. The Wassermann test was negative, and the colloidal gold curve was normal. The blood count showed 10,000 leukocytes; the stained blood smear was in the main normal. During the period of hospitalization, six treatments with artificial fever were given. This therapy was administered for a total of fifteen hours by means of the Kettering hypertherm at from 104 to 105 F. I saw the patient two days after his second treatment and observed a slight subsidence of the swelling of the disk in each eye. Vision in the right eye was considerably improved; the patient could see to get around unaided.

When he reported at my office on February 2, the vision in the right eye was 1/70; that in the left eye was 1/100. Projection of light was normal. At this time the disks exhibited marked atrophy, although the nasal border of the left disk still showed 1 diopter of elevation. The next and final observation of the patient was made on May 28. The vision in the right eye was 3/200, and in the left, 1.5/200. Each pupil measured 6 mm. and reacted sluggishly to light. There was typical postpapillitic atrophy of the nerve heads, and studies of the visual fields likewise gave typical results.

COMMENT

With respect to encephalitis that follows epidemic parotitis, practically nothing is known of the pathologic process. It is not known with certainty that the direct cause of mumps is either the direct or the sole cause of the neurologic phenomena. Nor may it be stated, except theoretically, that mumps activates a virus which is already existent in the central nervous system, thereby causing neurologic complications. In such a case, in the absence of infectious processes other than mumps, the encephalitis is naturally regarded as a complication of the primary disease.

From a review of the published accounts of cases of mumps in which there was ocular involvement, it appears obvious that the various treatments used have been regarded as of only slight, if any, real benefit. I have attempted to rationalize the treatments that seemed to warrant consideration in such cases. Theoretically, at least, any and all treatment should have as its objective a threefold purpose: first, the combating of toxins in the affected area; second, the maintenance of the nutrition of the involved nerve tissue, and third, the dehydration of the inflammatory zones without destruction.

Drainage of the spinal fluid has been recommended by Hubbard [3] to aid in decompressing the involved nerve tissue. This measure is universally recognized and is regularly practiced in cases of encephalitis. Hyland [8] inferred that the complications of mumps may be prevented by the use of convalescent serum. This prophylaxis is justified on the basis of acquired antibodies which combat the toxins of the disease. It may well be remembered, too, in this connection that human blood serum is a dehydrating agent. As such, it is amenable to control within certain limitations by the regulation of the dosage and by the interval between treatments. It can be seen from the case reported that fever therapy acted quickly in dehydrating the swollen optic nerves. In favor of this treatment, too, is the improved nutrition which should result from maintenance of the blood supply through the increased body temperature.

CONCLUSIONS

One cannot postulate in any case of encephalitis the improvement that may be anticipated by therapy any more than one can forecast the result that may follow the natural healing and compensatory phases of acute inflammations. Since it has won the favor of the medical profession at large, drainage of the spinal fluid stands and will have to stand on its merits as a therapeutic measure in cases of encephalitis following parotitis. Even though one accepts the premise that the antibodies of convalescent serum prevent the complications of mumps, there is no evidence to show that convalescent serum will aid in relieving established complications in the central nervous system. I mention this treatment merely as a clinical experiment of possible but unproved value. There is no denying the fact, however, that the fever therapy referred to in this case had a direct and beneficial effect on the patient.

8. Hyland, C. M.: Nebraska M. J. **22**:342, 1937.

SIMPLIFICATION OF THE O'CONNOR CINCH OPERATION

M. E. SMUKLER, M.D.
PHILADELPHIA

The O'Connor cinch operation for tendon and muscle shortening, described in a recent textbook as "not so easy to perform," is greatly simplified and shortened with the new instruments and operative procedure described here. It is my operation of choice in all cases of divergent squint and in some properly selected cases of convergent squint of moderate degree.

The tendon and muscle are divided into three or four equal strands from the insertion back toward the equator, one, two or three loops of a cable (Dr. O'Connor's term is "shortener") composed of four, six or eight strands of medium dermal suture being used. The number of muscle divisions, loops and cable strands depend on the age of the patient, the degree of squint and the condition of the tendon and muscle found at the time of operation. Slight overcorrection in each eye is desirable. Care must be taken to avoid increased tension of the tendon, caused by having the loops too near the cornea. This may result in keratitis or sloughing of the muscle. I have never encountered these complications, but they have been observed (Zentmayer).

INSTRUMENTS

The following instruments are required:

1. Cables of four, six or eight strands of medium dermal suture, 12 inches (30 cm.) long (fig. 1). The ends of these strands for 1¼ inches (3 cm.) are joined with several layers of metallic X cement. This united flexible metallic end is easily bent, forming a curved needle and needle holder. It enables the cable to be slipped easily under and around the strands of the elevated and divided muscle, so that they may be looped as often as is necessary. The other end of the cable is held together with some of the cement, so as to facilitate handling. The idea of the cable was devised by Dr. O'Connor, who has sent me a cable with only one end fastened together, and that to the extent of but ½ inch (1.3 cm.). However, the cable as constructed by me greatly simplifies the operative procedure.

Read before the Eye Section of the Philadelphia County Medical Society, April 7, 1938.

2. A sharp-tipped curved hook. This is used to separate the tendon and muscle into strands of equal width (fig. 2 *A*).

3. A blunt-tipped curved hook (fig. 2 *B*). This facilitates handling the strands of tendon and muscle and in separating the strands of muscle.

4. A pair of large flat muscle hooks (fig. 3). These hold the tendon and muscle well spread out.

5. A pair of three and four strand separators (fig. 4). One separator placed at each end of the divided tendon and muscle separates the strands and slightly elevates them from the globe, facilitating looping the cable around the strands as often as desired.

OPERATIVE PROCEDURE

1. A semicircular conjunctival incision is made, well in front of the insertion and extending backward parallel slightly above and below the margins of the tendon.

2. The tendon and muscle from the insertion back toward the equator are exposed and isolated. It is important to avoid extended mutilations and unnecessary pocket dissections. The steps of the new operative procedure follow: The fascia surrounding the tendon and muscle to be included in the strands is gently cleaned. This procedure facilitates the division of the tendon and muscle into strands of equal size; the insertion and removal of the separator; the wrapping of cable loops around the strands, pushing the loops together, and the removal of the cable at the end of fourteen days. It also gives a good adhering surface and stirs up a certain amount of reaction, which causes better union during the process of repair.

3. The flat muscle hooks are inserted at each end to hold tendon and muscle well spread out, so that they can be divided into three or four equal strands; a sharp curved hook is then inserted beneath, a small longitudinal incision or nick being made to separate the fibers. With the blunt hook inserted into the separation, it is extended from the insertion backward toward the equator for a distance slightly greater than the amount of shortening desired. Great care should be taken to avoid cutting across any muscle fibers; they should merely be separated. If the tendon and muscle are to be divided into three equal parts, this is best accomplished by inserting the sharp curved hook beneath each margin. If the division is to be into four strands, the tendon and muscle are divided into half from beneath. Each half is then divided into two strands by passing the sharp curved hook beneath each half at the upper and the lower tendinomuscular margin.

4. After the tendon and muscle have been divided into strands, one muscle hook is removed and replaced by a separator, inserted like a

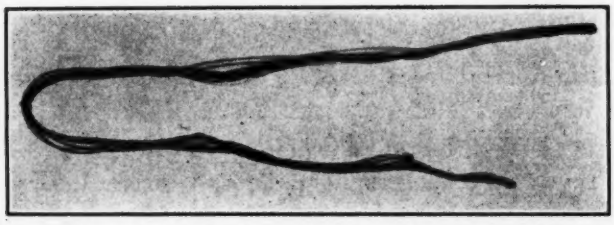


Fig. 1.—Cable of eight strands of medium dermal suture with metallic X cement at the ends.

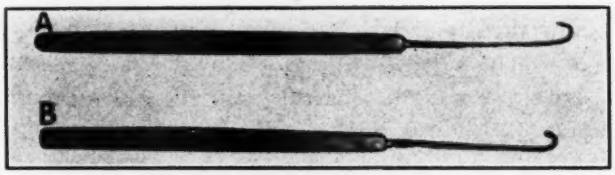


Fig. 2.—*A*, sharp-tipped curved hook to separate the muscle and tendon into strands of equal width. *B*, blunt-tipped curved hook to facilitate handling the strands of tendon and muscle. (One-half actual size.)

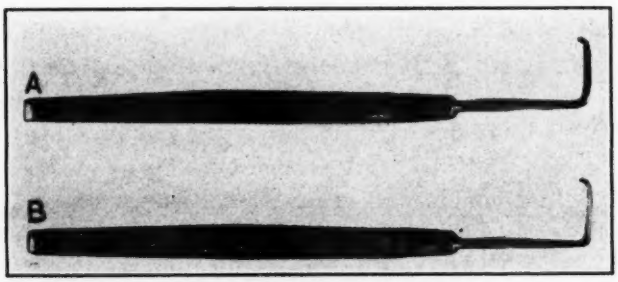


Fig. 3.—Pair of large flat muscle hooks, to hold tendon and muscle well spread out. (One-half actual size.)

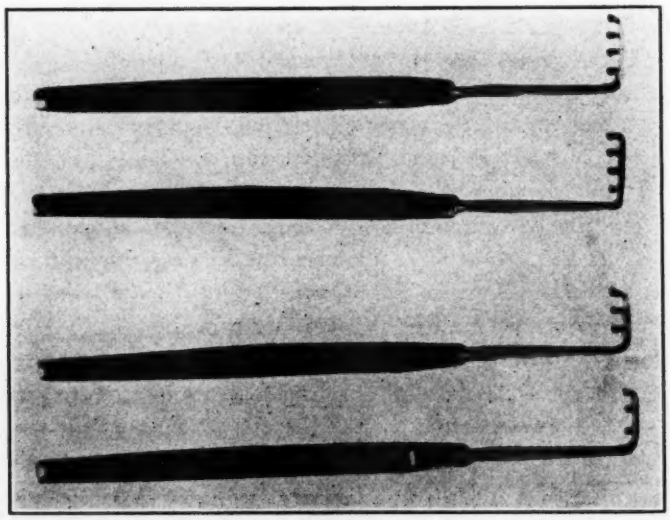


Fig. 4.—Pair of three and four strand separators. One at each end of the divided tendon and muscle separates the strands of tendon and muscle and slightly elevates them from the globe, facilitating looping the cable around the strands as often as possible.

muscle hook. Each strand will usually fall into a separate groove if the instrument is properly inserted and the tendon and muscle are properly prepared for its reception. If a strand does not fall in its proper place on the separator, it should be gently lifted with the blunt hook and properly placed. With the strands in place, the other muscle hook is removed and replaced by the other separator. This is inserted next to the first separator, so that the strands will be in their proper

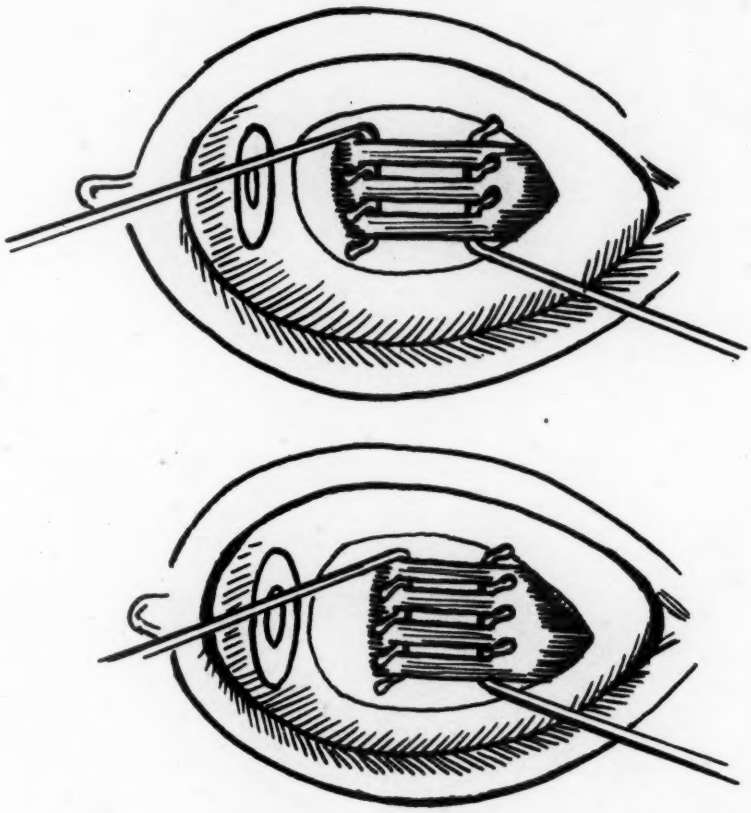


Fig. 5.—Three and four strand separators holding the strands apart and slightly elevated from the globe.

grooves. Then the second separator is moved to the other end of the divided muscle.

5. After the strands of tendon and muscle have been separated and slightly elevated from the globe, the separators are held in place at each end of the divided muscle by the assistant (fig. 5). The metal cable end (previously bent as desired) is now easily looped around the individual strands as often as is desired. When the looping on each strand is completed, the separators are removed (fig. 6). They come

out easily by reversing the procedure of insertion. The handle is turned down, then gently pushed downward and backward. This disengages the small uprights between the strands, and the instruments slide out from below the muscle, in the same manner as a muscle hook comes out. This procedure, complicated though it sounds, is simplicity itself in performance.

6. After the looping is finished on each strand and the separators are removed, the cable is pulled taut, and a tuck is completed. The

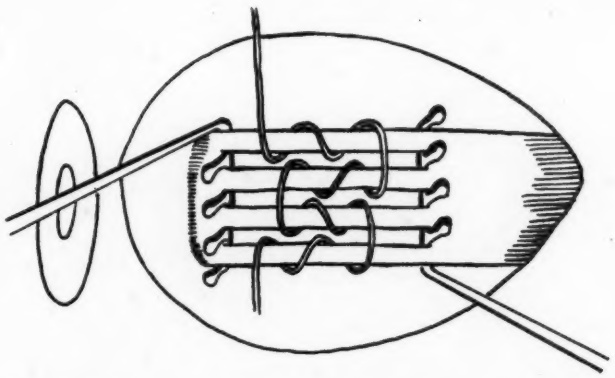


Fig. 6.—Diagram showing the cable double-looped around four strands of muscle, with the separators in place. These are removed when the looping is completed.

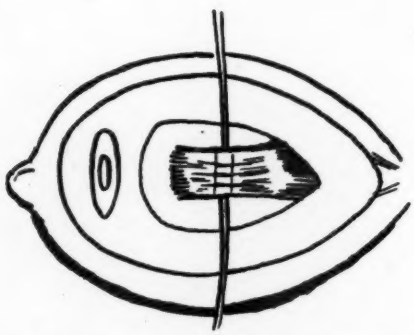


Fig. 7.—Diagram showing the cable pulled taut and the tuck completed.

looped strands of tendon and muscle are now pushed together by pressing the blunt hook at the upper margin of the muscle against a pair of forceps holding the cable at the lower margin (fig. 7).

7. The cable is cut 5 mm. above the upper margin of the tendon and 15 mm. below the lower margin. The conjunctival incision is closed with two or three silk sutures. The upper end of the shortener is buried under the conjunctiva. The lower end of the cable emerges from the incision to lie free in the sulcus.

8. The eyes are bandaged. The bandages are removed at the end of forty-eight hours, the silk sutures at the end of eight days and the cables at the end of fourteen days. It has been my experience that if the tendon and muscle have been properly prepared and the cable properly inserted around and between the strands, the cable slips out easily without the need of any local anesthetic.[1]

DISCUSSION

DR. L. F. APPLEMAN: Dr. Smukler has given what I believe to be some valuable aids in performing the O'Connor operation. First, in fastening both ends of the strands by metallic cement one provides a sufficiently stiff end to weave among the strands of muscle without the aid of a special needle, which is rather difficult to thread with six or eight strands of dermal suture, and at the same time the distal ends are held together; consequently, they will not whip all over the field of operation during insertion.

Again, the use of hooks which will hold each strand of muscle separated from the other, and especially the pair of separators held at either end of the exposed muscle, will greatly simplify the passage of the strands of dermal suture between and around them.

I should consider these hooks a great help and not more difficult to disengage than the plain hook which is generally used. In my experience, the greatest trouble was encountered in keeping each strand of muscle separated, after it was longitudinally divided by the sharp hook, until the others were separated. Almost always the dividing line was lost while the next strand was being found. This can be overcome now by the use of the small projection on the hook.

I like the O'Connor operation, especially in cases of strabismus of from 10 to 15 degrees, and particularly in correcting vertical deviations. The operation is advantageous for the following reasons: There is little reaction; no unsightly lumps occur under the conjunctiva, which if present would be months in disappearing; the period of hospitalization is short, the patients being able to go home in two or three days, and the divided ends of the cable give little discomfort, as they lie well up in the cul-de-sacs and are normally removed at the end of two weeks simply by pulling them out a few at a time, or singly, without any discomfort.

I think that Dr. Smukler has done a service in devising these means of simplifying the technic.

1940 North Broad Street.

1. O'Connor, R.: The Cinch Shortening Loop in Surgery of the Extra-Ocular Muscles, West. J. Surg. **39**:670 (Sept.) 1931.

PACKING OF INTERNAL CAROTID ARTERY WITH MUSCLE IN TREATMENT OF CAROTID-CAVERNOUS ARTERIOVENOUS ANEURYSM

E. S. GURDJIAN, M.D.
DETROIT

The management of carotid-cavernous arteriovenous aneurysm has received extensive discussion in the literature.[1] Various methods of treatment which have been used, some with greater success than others, are as follows: (1) ligation of the internal carotid artery or of the internal carotid artery and the common carotid artery on the affected side; (2) preliminary ligation of the internal carotid artery followed by intracranial ligation of the same artery;[2] (3) ligation of the veins in the orbit; (4) a direct attack on the apex of the orbit; (5) plugging of the fistula in the artery with muscle introduced into the blood stream of the internal carotid artery,[3] and (6) injection of various substances with a view to helping clotting and eventual closure of the fistula in the affected artery and in other arteries. Treatment of this condition by ligation of the affected internal carotid artery has been found to be the most successful procedure.

The rationale of ligation of the carotid artery is to cause the formation of a blood clot which will eventually obliterate the fistula in the cavernous sinus. That this may be brought about by ligation of the internal carotid artery, or better still, by ligation of both the internal and the external carotid artery on the affected side, is well attested to by the excellent results obtained in fully 50 per cent of the cases.

In the case to be reported preliminary ligation of the common carotid artery at the junction of its internal and external branches on the right side proved to be a failure. The possibility of introducing small bits of muscle tissue into the blood stream of the internal carotid artery was considered. However, to insure against embolic phenomena it was thought more advisable to introduce a strip of muscle into the internal carotid artery and to plug the artery as high as possible by pushing the muscle toward the cranial cavity with an appropriate instru-

1. Locke, C. E.: Intracranial Arteriovenous Aneurysm or Pulsating Exophthalmos, Ann. Surg. **80**:1, 1924.

2. Dandy, W. E.: Treatment of Carotid Cavernous Arteriovenous Aneurysm, Ann. Surg. **102**:916, 1935.

3. Hamby, W. B., and Gardner, J. W.: Treatment of Pulsating Exophthalmos: Report of Two Cases, Arch. Surg. **27**:676 (Oct.) 1933.

ment. I felt that by this method there was little likelihood of bits of muscle breaking off and entering the venous circulation of the sinus. The results were so excellent that the case is presented as an argument in favor of primary plugging of the internal carotid artery with muscle, after one is sure that this can be done without cerebral complications. With this method a much shorter distance need be covered by the clot in order to effect eventual closure of the fistula in the artery. It is also reasonable to assume that the percentage of cures would be greater. Of course, plugging of the internal carotid artery with muscle is no different than ligation of the artery and only slightly more complicated.

REPORT OF A CASE

History.—M. B., a white man, entered the Grace Hospital on April 7, 1936, complaining of nausea, vomiting, swelling of the eyes and inability to elevate the upper lids. About a week before admission he suddenly became nauseated and vomited several times. The following morning he had a severe headache across the forehead. He complained of diplopia, and there was beginning swelling of the right eye. Two days later there was marked swelling of the right eye with inability to elevate the upper lid. Pulsations were felt in the head. In another three days there was swelling of the left eye.

Examination.—Twelve days after the onset of the condition examination showed marked eversion of the conjunctivas, especially the right; inability to elevate the upper lid of either eye, and inability to move either eyeball in any direction. There was slight dilatation of the pupil on the right side, with practically complete blindness. With a stethoscope, pulsations could be heard over the right eye, the forehead and the temporal region. There were no pulsations on the left side.

A diagnosis was made of spontaneous carotid-cavernous arteriovenous aneurysm on the right side.

Course.—For the next eight days digital pressure was applied to the right common carotid artery to obliterate it. At the beginning the pressure was applied for three minutes; the period was then increased, until on the eighth day the patient was able to stand half an hour of pressure without untoward symptoms. When this was accomplished he was ready for ligation of the internal and external carotid arteries.

Operation.—On April 17 the right common carotid artery and the internal and external carotid arteries were exposed, local anesthesia being used. A removable clamp was applied on the common carotid artery for forty-five minutes, with no untoward symptoms. The artery was then ligated at the junction of its two terminal branches.

There was marked diminution in the pressure of both orbits. The patient continued to show marked improvement in the left eye and moderate improvement in the right eye, and there was practically complete cessation of the pulsations in the head. He was discharged on April 27.

Second Admission.—He reentered the hospital on May 12, because of a return of practically all of the symptoms complained of at the first admission. There was a return of exophthalmos and marked eversion of the conjunctivas, so that the eyeballs could hardly be seen when the upper lids were elevated.

Second Operation.—On May 13 the carotid complex was reexposed on the right side. The junction of the common carotid artery and the external and internal carotid arteries was found to be completely thrombosed. Above this level the internal carotid artery was soft and evidently contained fluid blood. A rubber clamp was applied on the internal carotid artery, and a longitudinal incision 2 cm. in length was made. The index finger of the left hand was passed under the artery, and by pulling the finger toward the surface of the body the arterial lumen was easily obliterated. A previously obtained strip of muscle about 20 cm. long and 0.25 cm. in diameter was introduced into the lumen of the artery and

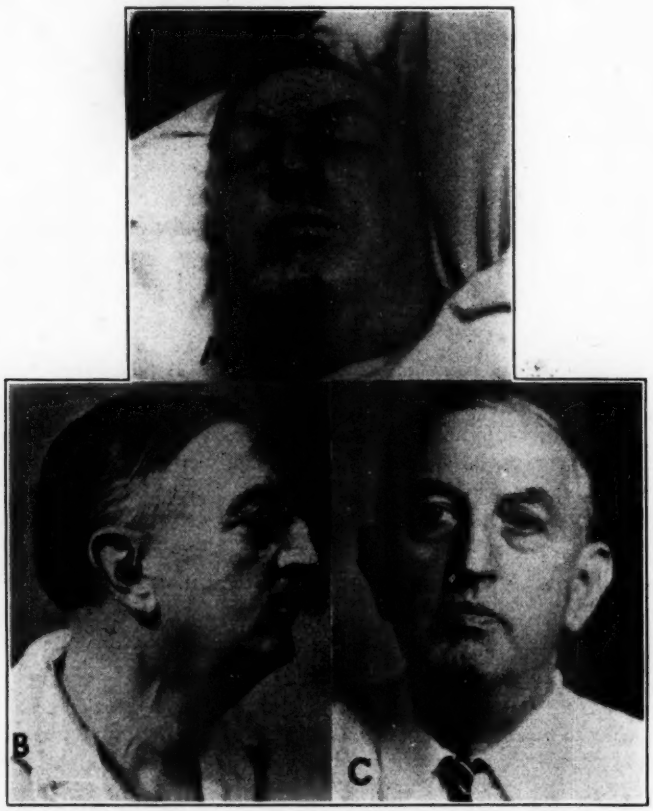


Fig. 1.—*A,* photograph of the patient on admission to the hospital, at which time he had bilateral exophthalmos, bilateral ptosis, complete paralysis of all extraocular muscles and practically complete blindness of the right eye. *B* and *C.,* photographs of the patient fourteen months later. Complete return of the movements of the extraocular muscles, 50 per cent return of vision in the right eye, and complete disappearance of the pulsations in the head had taken place.

pushed upward with a probe. By this process the internal carotid artery became completely plugged up to the base of the skull. A ligature was then applied at the point of introduction of muscle.

Course.—The course in the hospital after the second operative procedure was uneventful. There was marked improvement in each eye. The bilateral exoph-

thalmos receded practically completely within a week. In two weeks the patient was able to elevate the upper lids. In three months the movements of the extraocular muscles returned completely, and there was 50 per cent return of vision in the right eye. Photographs made on year and two months later showed an excellent result.

COMMENT

If the physician is satisfied that ligation of the carotid artery is the procedure of choice in the treatment of a given carotid-cavernous arteriovenous aneurysm, plugging of the internal carotid artery with

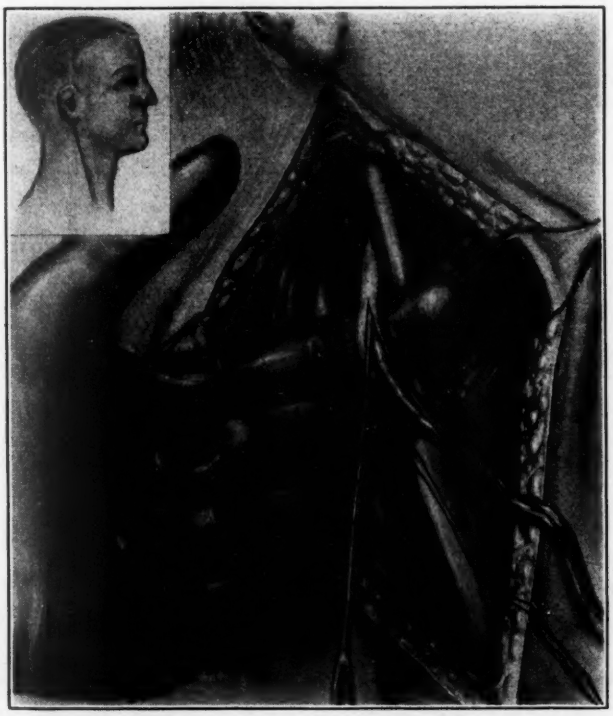


Fig. 2.—Diagrammatic representation of the technic for packing the internal carotid artery with muscle.

muscle seems a more reasonable procedure than mere ligation. The plug may easily be pushed as high as the base of the skull; hence a much shorter distance is left for further clotting in order to obstruct the fistula in the artery. The procedure is practically as simple as ligation of the carotid artery. A strip of muscle about 20 cm. long and 0.5 cm. in thickness may be obtained from the exposed sternocleidomastoid muscle. After exposure of the carotid complex, a clamp is applied below and above the area of incision of the internal carotid artery. The artery is then incised longitudinally, and the lumen is exposed. After the index finger of the left hand is passed under the incised artery,

the upper clamp is removed, and the bleeding is controlled by pressure from the index finger looping the artery. The strip of muscle is then pushed into the lumen with an appropriate probe. Every time the probe is removed to start the plugging procedure over again, the pressure by the index finger controls all the bleeding. On completion of the procedure the artery is ligated above and below the point of introduction of muscle.

SUMMARY

Packing of the internal carotid artery with muscle as high as possible affords a better opportunity for the cure of carotid-cavernous arteriovenous aneurysm. With this method a much less extensive blood clot is needed above the level of the muscle plug to effect eventual closure of the fistula in the artery. On theoretic grounds, at least, plugging of the internal carotid artery with muscle should be much more efficient in the treatment of this condition than mere ligation of the artery.

MAGNESIUM CONTENT OF CAPSULATED LENSES

A REVIEW OF ITS PROBABLE IMPORT; PRELIMINARY REPORT

ISADORE GIVNER, M.D.
AND
CATHERINE F. GANNON, M.A.
NEW YORK

The constantly increasing therapeutic use of magnesium salts, particularly in the United States, makes one consider whether or not harmful effects on the eye can be produced. Burge [1] had shown that the magnesium content of cataracts removed in this country was five times that of cataracts removed in India.

The possibility of a relation of intake of magnesium to pathologic changes in the lens was suggested by the following case:

R. K., a housewife aged 49, was first seen on Dec. 4, 1935, complaining of failing vision in each eye for the past year.

Examination disclosed soft mature cataracts, with good light projection. A search into all possible etiologic factors, which were present at the time or which may have existed in the formative period, failed to disclose any of a dietary, hereditary, metabolic, endocrinologic or toxic nature.

Three years previously, at a time when from the patient's point of view normal vision existed except for myopia which was corrected adequately by glasses, she experienced a digestive upset which caused her to seek the aid of her family physician. Magnesium oxide was prescribed to be taken three times daily. The immediate symptoms disappeared, but the patient continued to take the medicament as prescribed, unknown to her physician, until the day she presented herself for ophthalmologic examination. The possibility of a relation of the medication to the formation of cataract was uppermost in her mind, but at that time a conclusive opinion could not be given concerning this.

The amount of experimentation done to relate changes in the calcium content of the body to formation of cataract justifies some consideration of the relation of magnesium to calcium.

In reviewing the more outstanding data on the former, one finds the following observations:

1. Although the calcium content of bone as determined experimentally, the calcium content of the blood and the calcium metabolism

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From the Ophthalmological Service of Dr. Webb W. Weeks, the Bellevue Hospital.

1. Burge, W. E.: Arch. Ophth. **38**:435, 1909.

of patients with senile cataract are normal, the calcium content of the cataractous lens increases from an almost negligible quantity in the normal lens to 12.5 per cent (Berger). Mackay and his co-workers,[2] using 16 normal lenses, found 1.01 per cent of calcium ash as compared with 5.49 per cent in 16 lenses with senile cataract. Because of the greatly increased calcium content, they suggested that some special relation exists between calcium and the development of cataract.

2. In a study of the permeability of the capsule of the lens, Friedenwald [3] found it higher than that of other biologic membranes, exceeding that of capillaries. Although it has not been definitely proved, he expressed the opinion that a decrease in capsular permeability in vivo leads to the formation of cataract. Exposure of the capsule to calcium chloride, potassium cyanide and egg albumin caused a reduction in the permeability.

3. Kirby [4] found slight reductions in calcium toxic to a culture of epithelial cells of the lens, but an increase of calcium had no effect.

4. Burge [5] and Hinrichs [6] found that lenticular opacities were more readily produced in lenses by ultraviolet irradiation in the presence of calcium salts. This has not been sufficiently verified.

In an effort to point out the antagonistic relation of magnesium and calcium, the following observations may be cited:

1. A lowering of calcium produces a slight increase in magnesium. Equally true is the fact that administration of magnesium increases the elimination of calcium. On the other hand, anesthesia produced by intravenous or subcutaneous injection of magnesium chloride is counteracted by calcium salts.

2. A diminution of protein or an augmentation of phosphates results in a reduction of the calcium content of serum, whereas the magnesium content is unaffected.

3. Carswell and Winter [7] expressed the belief that with an adequate intake of phosphorus administration of magnesium favors storage of calcium.

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2. Mackay, G.; Stewart, C. P., and Robertson, J. D.: Brit. J. Ophth. **16**: 193, 1932.

3. Friedenwald, J. S.: The Permeability of the Lens Capsule, with Special Reference to the Etiology of Senile Cataract, Arch. Ophth. **3**:182 (Feb.) 1930; The Permeability of the Lens Capsule to Water, Dextrose and Other Sugars, ibid. **4**:350 (Sept.) 1930.

4. Kirby, D. B.: Permeability of the Lens Capsule, Arch. Ophth. **5**:856 (June) 1931.

5. Burge, W. E.: Am. J. Physiol. **36**:21, 1914.

6. Hinrichs, M. A.: Proc. Soc. Exper. Biol. & Med. **27**:535, 1930.

7. Carswell, H. E., and Winter, J. E.: J. Biol. Chem. **93**:411, 1931.

4. Parathyroid extract causes a loss of magnesium as well as of calcium, while parathyroidectomy is followed by a retention of magnesium.

5. Swingle and Wenner [8] stated that although calcium and magnesium do not subserve the same functions within the organism they can be substituted for one another in the alimentary mixture. They found that parathyroidectomized animals could be preserved from tetany by the addition of magnesium salts to diets which would not of themselves prevent tetany if the salts were given before the calcium content of the serum had fallen. When the calcium content was low, however, magnesium salts did not prevent tetany. It was inferred that magnesium was substituted for the calcium in the excretion and that by that means absorption of calcium was promoted.

6. Muscle normally contains 21 mg. of magnesium per hundred grams of tissue, while only 7 Gm. of calcium is found in a like quantity of muscle. Bone contains one eighth as much magnesium as calcium. In the blood, calcium is found almost exclusively in the serum, while magnesium is equally distributed between serum and cells, if anything a slight excess being found in the corpuscles. Normally, from 2 to 3 mg. of magnesium per hundred cubic centimeters of serum against from 9 to 11 mg. of calcium is found. In whole blood cells the magnesium content is found to be from 2.3 to 4 mg. per hundred cubic centimeters of whole blood cells. In spinal fluid from 3 to 3.5 mg. of magnesium per hundred cubic centimeters is normally found. Duke-Elder,[9] using intraocular fluids of the horse, found 100 cc. of aqueous to contain 0.0062 Gm. of calcium as compared with 0.0026 Gm. of magnesium, whereas in the vitreous he found 0.0068 Gm. of calcium as compared with 0.0020 Gm. of magnesium.

Fischer [10] in 1933 by colorimetric measure found magnesium to be present in greater quantities than calcium in the normal lens and ciliary body of the calf, cow and pig, calcium being found in greater quantities than magnesium in the cornea, vitreous and sclera.

A still less understood relation exists between magnesium and sugar. Magnesium is a part of chlorophyll. The latter's part in carbohydrate building is now being thoroughly investigated. Lohmann has found that the retina has a higher percentage of sugar than other adult tissues and that magnesium "fait partie du coferment, de la glycolyse." Wolff and Bourquard,[11] studying the distribution of magnesium in the tissues of the eye, found that the retina had the highest magnesium content next to muscle tissue. They did not report on the magnesium in the lens.

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8. Swingle, W. W., and Wenner, W. F.: Proc. Soc. Exper. Biol. & Med. **23**:432, 1926.

9. Duke-Elder, W. S.: Text Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1933, vol. 1, p. 427.

10. Fischer, F. P.: Arch. f. Augenh. **107**:295, 1933.

11. Wolff, R., and Bourquard, A.: Compt. rend. Soc. de biol. **124**:319, 1937.

While glycosuria following the injection of magnesium sulfate was casually noted by Meltzer and Auer, the first careful study of the effect of magnesium in this direction was carried out by Underhill and Closson.[12] In the estimation of these workers the hyperglycemic action of magnesium is the result of a disturbance in respiratory action. Lang and Rigo injected from 25 to 100 mg. of a magnesium salt per kilogram of body weight and increased the dextrose concentration of the blood from 10 to 30 per cent. They assumed that the hyperglycemia is produced by the action of magnesium on the sympathetic nervous system.

A study of the relation of sugar to lens metabolism brings forth the following data:

1. Human lens contains 60 mg. of free dextrose per hundred cubic centimeters of lens (Lottrup-Andersen [13]).

2. The lens utilizes sugar.

3. Kronfeld and Bothman [14] found lens sugar to be the same as blood sugar and reported that it was a hexose.

4. The capsule of the lens is highly permeable to dextrose and less so to sucrose and galactose.

5. Insulin increases the permeability of tissue.

6. The ratio of the sugar in the blood to that in the aqueous is 0.82, the sugar in the aqueous increasing when that in the blood increases. (Duke-Elder has held that cataract in association with diabetes is the result of abnormal fluid traffic.)

EXPERIMENTAL OBSERVATIONS

In this preliminary report on magnesium and cataract the first approach was in the nature of communications to mines where magnesite was worked to find out whether or not men who were daily in contact with magnesium had complained of visual disturbances. The replies were all in the negative.

Next a determination of the magnesium content of the blood serum of twenty-seven patients with cataract was undertaken by the Cohen-Briggs method.[15] The highest recorded magnesium content in this group was 5.1 mg. per hundred cubic centimeters of serum, and the lowest value was 2.1 mg.

The patient whose condition stimulated this investigation had 4.8 mg. of magnesium per hundred cubic centimeters of serum (and after

12. Underhill, F. P., and Closson, O. E.: Am. J. Physiol. **15**:321, 1905.
13. Lottrup-Andersen, C.: Acta ophth. **5**:226, 1927.
14. Kronfeld, P. C., and Bothman, L.: Ztschr. f. Augenh. **65**:41, 1928.
15. Cohen, H.: Quart. J. Med. **20**:173, 1927.

taking no magnesium for five weeks, 4.5 mg.). The average reading was 3.3 mg. (Normally, the blood serum contains from 2 to 3 mg. of magnesium per hundred cubic centimeters of serum.)

Spaeth [16] reported signs of tetany in a case of dinitrophenol cataract postoperatively. An opportunity was had to study the blood of a patient poisoned with dinitrophenol while acutely ill and showing jaundice. The magnesium content of the blood serum was 2.5 mg.

In 21 of the foregoing cases the calcium content of the blood serum was likewise determined. The lowest content was 9.2 mg. and the highest 11.5 mg. per hundred cubic centimeters (normal, from 9 to 11 mg.). The average content was 10.4 mg.

There was no correlation between the amount of magnesium and that of calcium. In cases in which both 2.1 and 5 mg. of magnesium were found the calcium content was 11 mg. per hundred cubic centimeters of serum.

Next the magnesium content of cataractous lenses removed in capsule at operation was determined. The method used was as follows:

The ash of a single lens was dissolved in 10 cc. of approximately tenth-normal hydrochloric acid in a centrifuge tube. Saturated ammonium oxalate was added, and the procedure was then carried out as for serum.[15]

Standard solutions containing 1, 2, 3, 4 and 5 cc. of Briggs' stock standard (containing 0.007 mg. of magnesium per cubic centimeter) should be made up for close comparison in the colorimeter. (When less than 5 cc. is used, one should make up to 5 cc. with hundredth-normal sulfuric acid.)

Eighteen lenses were analyzed. One removed at autopsy from a normal eye of a patient who died of pulmonary tuberculosis showed 0.014 mg. of magnesium in the entire lens.

For the cataractous lenses, the findings ranged from 0.006 to 0.028 mg. of magnesium, the average being 0.0163 mg. In the group were 2 black cataracts containing 0.010 and 0.028 mg. of magnesium, respectively.

From this analysis there appeared to be no variation from the normal magnesium content of lenses.

The lens of the patient taking magnesium oxide, removed by the extracapsular route, showed less than 0.0035 mg. of magnesium. This finding made necessary the explanation that possibly the capsule with the cortex that adhered to it must contain as much as if not more magnesium than was present in the expressed portion. Accordingly, 7 lenses removed intracapsularly were stripped of their capsules, and these together with some underlying cortex were placed in a porcelain crucible. In another crucible were placed the bulk of the lenses.

16. Spaeth, E. B.: Am. J. Ophth. **19**:320, 1936.

Determinations of the magnesium showed the following values: (*a*) 0.062 mg. in 7 capsules and adherent cortex and (*b*) 0.058 mg. in 7 nuclei.

To determine experimentally what effect magnesium taken orally could produce in the lens, two experiments were undertaken.

Patients with immature cataract which interfered appreciably with vision were given magnesium oxide to see if there was any appreciable acceleration in the maturing process. This was carried out in 10 cases. Observation over a year failed to disclose any changes that seemed to differ from the usual progress in such cases. None of the cataracts retrogressed; 6 showed no change during the period of observation.

The other method, which allowed for more accurate notation, consisted of feeding 3 white mice, 6 weeks old, of the same litter a diet that would not of itself produce cataract. One mouse was given 1 cc. of magnesium chloride (0.02 Gm. of magnesium) daily by mouth with an eye dropper. A second mouse was given twice this quantity of magnesium chloride, 2cc. (0.04 Gm. of magnesium). A third mouse of the same litter was not given any magnesium. This experiment was continued for eight months, after which the right eye of each rat was removed. Chemical analyses of the lenses showed the following data: The entire lens of rat A, fed no magnesium, contained 0.0038 mg. of magnesium; that of rat B, fed the smallest quantity of magnesium, 0.0043 mg., and that of rat C, fed the largest quantity of magnesium, 0.0062 mg.

The left eye was removed from each rat for histologic study and showed no evidence of cataract formation. The ciliary body, which normally contains a higher amount of magnesium, was likewise [illegible]logically normal.

SUMMARY

Blood serum of patients with normal lenses compared with the blood serum of patients with cataracts showed no appreciable difference in the magnesium content.

The amount of magnesium in the cataractous lens was not found to differ appreciably from that of a normal lens.

The amount of magnesium in the capsule and adherent cortex is greater than that in the remaining nucleus of the lens.

Two rats fed daily with varied amounts of magnesium over eight months had a higher magnesium content in their lenses than a rat given no magnesium.

CONCLUSION

Whereas an increased magnesium intake might increase the amount of magnesium in a lens, from our present knowledge we cannot infer any relation between intake of magnesium and formation of cataract.

A CLASSIFICATION OF CONCOMITANT STRABISMUS

RESULTS SECURED IN VARIOUS TYPES

GEORGE P. GUIBOR, M.D.
CHICAGO

A report of the possibilities of orthoptic training was presented before the Section on Ophthalmology of the American Medical Association in June 1933. A second report on the practical details in the orthoptic treatment of strabismus was presented in June 1934. These reports were based on observations made on two groups of patients. One, a control group at the Children's Memorial Hospital, received no orthoptic training, atropinization, occlusion of the fixing eye and a correction of the refractive errors being the only measures carried out. The second group, those seen at Northwestern University, were given orthoptic training. This report is a discussion of these two groups and of additional patients who were sent in for observation at the Children's Memorial Hospital and for observation and treatment at Northwestern University; every subject considered has been under observation for six months or longer.

The examination of the patients included: (1) determination of vision, with and without glasses; (2) determination of muscle balance, motility, the angle gamma and the angle of squint, and (3) determination of the degree of fusion.

The following classification of types of strabismus has been used for two years and has been of help in differentiating patients who will be aided materially by nonsurgical measures from those who must have operative correction.

1. Pseudoparalytic strabismus
2. Accommodative strabismus
3. Strabismus due to or associated with fusion defects
4. Strabismus due to or associated with amblyopia
5. Strabismus due to or associated with neuromuscular defects
6. Strabismus due to or associated with anisometropia
7. Strabismus due to or associated with multiple defects

This classification is not entirely original. Consultation of the works of the authors cited will verify this, though no one author has included

From the Departments of Ophthalmology, Children's Memorial Hospital, and Northwestern University Medical School.

all the salient points given in this outline. If squint is recognized as a symptom complex with multiple causes, to be treated by multiple therapeutic methods, the problem of diagnosis and treatment is not so difficult.

The classification of concomitant squint has been attempted by many authorities. Worth [1] discussed motility defects, especially subnormal external rotation, which occurred in 19 per cent of 1,523 cases, and emphasized the undue shortness of the internal rectus muscles. His conclusions were that such defects are secondary and not the cause of squint.

Worth stated, however, that even though the essential cause of squint is a defective fusion ability, secondary or exciting causes exist, such as hyperopia, anisometropia, motor defects, amblyopia, mental disturbances, hereditary factors, birth injuries and specific fevers. He was aware therefore that secondary causes exist and are important in the treatment of squint.

Sheila Mayou [2] classified divergent squint according to the refractive errors present; in a group of 93 cases of divergent squint there were 48 cases of emmetropia, 26 cases of hypermetropia and 17 cases of myopia. The general conclusion was that small deviations, of 10 or 15 degrees, are relieved by orthoptic training; those over 15 degrees are relieved by surgical treatment plus orthoptic training. The prognosis is best in those cases of squint associated with emmetropia and poorest in those associated with myopia.

Pugh [3] similarly classified a series of cases into instances of refractive error squint (62 per cent), fusion defect squint (15 per cent), psychologic squint (21 per cent) and physical defect squint (20 per cent).

The classification in this article was made independently but at the same time as those of the foregoing authors. I believe that a review of the tables presented will demonstrate this classification to be more complete than most classifications and will show those factors associated with squint which must be treated and overcome to produce recovery from strabismus.

Table 1 shows the relative value of tests for determining the types of squint. It can be seen that the diagnosis of the various types is made as follows: (1) pseudoparalytic squint, by simultaneous atropinization of each eye and determination of the motility; (2) accommodative squint, by atropinization of each eye, correction of the refractive error and measurement of the angle of squint for far and near vision, with and without glasses; (3) squint associated with fusion defects, by fusion

1. Worth, C. A.: Squint, London, Baillière, Tindall & Cox, 1921, p. 48.
2. Mayou, S.: Brit. J. Ophth. **19**:37 (Jan) 1935.
3. Pugh, M.: Brit. J. Ophth **18**:446 (Aug.) 1934; Squint Training, London, Oxford University Press, 1936.

tests and by three attempts to train the patient to fuse when such ability apparently is absent or defective; (4) the amblyopic type of squint, by determination of the visual acuity; (5) strabismus associated with muscular defects, by studying the motility and measuring the angle of squint and by the use of prisms for constant wear, especially if a vertical deviation is present, and (6) the anisometropic type of squint, by determination of the refractive error.

TABLE 1.—*Relative Value of Tests for Determining Common Types of Squint*

Tests	Pseudo-paralytic Squint	Accommodative Squint	Squint Associated with			
			Defects in Fusion	Amblyopia	Muscular Defects	Anisometropia
Test for visual acuity	.....	.....	.....	Poor vision in one eye	.....	.....
Test for motility	Apparent paralysis of the external rectus muscles	.....	.....	.....	Under-action and over action disclosed	.....
Atropinization...	Appearance of external rotation	Decrease in squint	.....	.....	.....	.....
Refraction.....	.....	Decrease in angle of squint	.....	.....	.....	Unequal refractive error in each eye
Determination of angle of squint...	.....	Less for distant than for near vision	.....	.....	Variable	.....
Fusion tests.....	.....	Some fusion ability	Marked fusion defects	Total partial suppression of amblyopic eye	.....	Fusion of large objects only
Therapeutic tests with fusion training	.....	Normal fusion produced	Improvement in fusion	.....	.....	.....
Prism trial test...	.....	.....	.....	.....	Possible decrease in horizontal deviation on correction of the vertical deviation	.....

After the diagnosis of the type of squint has been made, the refractive errors are corrected as follows:

In cases of amblyopic squint of not too severe a degree the correction before the fixing eye is decreased so that when it is atropinized the visual acuity of this eye is equal to, or less than, that of the amblyopic eye (table 2).

In cases of accommodative squint a full correction is prescribed for distant vision and a bifocal addition of a plus 3.00 D. sphere for near vision. Atropinization of both eyes is done for from one week to a month and can be repeated at will.

In cases of anisometropic squint with equal visual acuity in each eye attempts may be made to equalize the difference in the size of the retinal images, after the methods outlined by Ames. Nothing has been done with this method in the clinics at Northwestern University and at the Children's Memorial Hospital.

In the series of charts which I have prepared at Northwestern University the card for measuring the size of the retinal images, as suggested by Dr. Andrews, may be used to measure roughly the difference in the size of the retinal images.

DIFFERENT TYPES OF SQUINT

Pseudoparalytic Strabismus.—Dodds [4] and others have observed cases of pseudoparalytic squint. Dodds has compiled a bibliography covering most reports of cases of pseudoparalytic or congenital muscular

TABLE 2.—*Methods of Correcting Refractive Errors*

Amblyopic Type of Squint	Accommodative Type of Squint	Anisometropic Type of Squint
Amblyopic eye: Total correction Fixing eye: Corrected to equal visual acuity under atropinization of the amblyopic eye	For distant vision: Total correction of ametropia in each eye For near vision: Bifocal lenses +3.00 D. sph.	*Correction of difference in size by methods of Ames (if necessary)

squint during the last sixty years. He expressed the belief that the condition in such cases is true paralysis of the abducens nerve.

This condition is found in infants and apparently exists from birth. One eye converges, or both eyes converge simultaneously, and abduction ability seems reduced or absent. In the bilateral type both eyes appear immobile; the palpebral fissure is narrow, and there is an apparent enophthalmos. The diagnosis of bilateral paralysis of the abducens nerve is often made in these cases. When the eyes are atropinized, however, an alternating convergence of moderate severity (25 degrees) appears. Careful examination reveals the presence of almost normal abduction.

In cases of pseudoparalytic squint the prognosis regarding a cosmetic recovery by nonsurgical means is poor. Yet loss of visual acuity and the development of abnormal correspondence may be prevented by atropinization of the eyes.

The following case demonstrates this type of squint:

C. M., a girl aged 2 years, appeared in September 1933 at the Children's Memorial Hospital; she was of a family of 10 children, 1 other child being cross eyed. There was bilateral simultaneous convergence of 35 degrees. Each

4. Dodds, L.: Brit. J. Ophth. **10**:649 (Dec.) 1926.

eye was apparently fixed in this position. No abduction ability could be elicited. A diagnosis of bilateral paralysis of the abducens nerve was made by three examiners. A 0.5 per cent solution of atropine sulfate was instilled into each eye twice a day for several weeks. When cycloplegia was complete, the abduction ability appeared. An alternating convergence of 35 degrees gradually became one of 50 degrees and was associated with an alternating sursumvergence of 20 degrees.

In June 1934 under cycloplegia with atropine, the retinoscopic findings were:

R. 4.00 D. sph. ⌒ 2.00 cyl., axis 145
L. 5.25 D. sph. ⌒ 1.50 cyl., axis 135

The prescription for glasses was as follows:

R. 2.75 D. sph. ⌒ 2.00 cyl., axis 45
L. 4.00 D. sph. ⌒ 1.50 cyl., axis 135

In June 1936 there was alternating convergence of 55 degrees with an alternating sursumvergence of 20 degrees, with and without glasses, for far or near vision. There was a slight defect in the abduction of the right eye.

A diagnosis of pseudoparalytic squint was made. Though hyperopia was present, the squint was unaffected by correction of the refractive errors; therefore, this is not of the accommodative type.

ACCOMMODATIVE STRABISMUS

The characteristics of accommodative convergent squint are:

1. It is associated with hyperopia.

2. The angle of squint decreases or disappears (*a*) under atropinization, (*b*) with correction of the hyperopia and (*c*) with distance fixation.

3. This type of squint is often associated with a moderately severe amblyopia, which disappears rapidly when the fixing eye is occluded.

4. It is associated with good fusion ability.

The technic, therefore, in treating this type of strabismus is purely orthoptic, which is understood to include refraction, atropinization of one or both eyes, occlusion and fusion exercises. A complete correction of the hypermetropia (determined under cycloplegia with atropine) is prescribed. Bifocal lenses with an addition of a plus 3.00 D. sphere for near vision are prescribed to relax the spasm of accommodation for near vision which is so often present in these cases.

It is important that the bifocal segment should extend 2 mm. above the lower line of the lid. Stereoscopic ability is usually present in this type. Development of fusion amplitude and of abduction power is necessary to produce parallelism of the visual axis in cases of uncomplicated accommodative squint. In such cases the visual axes are parallel or almost parallel with glasses, but there is squint for near vision or when the glasses are removed.

Surgical technic at first glance might seem a satisfactory method for eliminating accommodative squint and also the use of unsightly convex

lenses. For the youthful subject the weakening of the internal rectus muscles by recessions might seem to produce no handicap or strain when the accommodation is powerful. But when the patient becomes more mature and a relaxation of accommodation and its associated relaxation of convergence occurs, a true convergence insufficiency or a divergent squint may occur.

STRABISMUS DUE TO OR ASSOCIATED WITH FUSION DEFECTS

Concomitant squint may be due to a fusion defect. Most patients with squint have some difficulty in fusion and may be unable to achieve it when first examined. Most of them, however, acquire the ability rapidly. Worth [5] expressed the belief that a defect in fusion is the primary cause of squint in most cases. On the other hand, Hansell and Reber [6] stated that this inability to fuse at first is a suppression rather than an absence of the fusion faculty.

In cases of alternating squint with little or no refractive error and with normal vision in each eye it seems likely that the faculty of fusion might be inferior to that of normal persons. That it is not entirely absent, however, is demonstrated by the patients with alternating squint who were seen at the orthoptic clinic at Northwestern University. Four of 7 patients have developed stereoscopic ability. Three have been unable to do so and have alternating monocular vision (pseudobinocularism).

With these facts in mind, a rigorous method of examination for fusion defects must be adopted before a diagnosis of defect or absence of fusion ability is made. For this purpose I have adopted the following criteria for the absence or defect of fusion ability: 1. Depth perception with the Howard apparatus is absent or much defective. 2. Stereoscopic ability with the amblyoscope, synoptophore and stereoscope is absent or defective after four trials, each trial extending over fifteen minutes.

With such a routine, few patients (15 per cent) are found with an absence of fusion faculty or a marked reduction in ability after several fusion exercises.

STRABISMUS DUE TO OR ASSOCIATED WITH AMBLYOPIA

It is certain that the term amblyopia is interpreted differently by different authors. Some believe that it is a mere difference in visual acuity between the two eyes, readily amenable to treatment. Others

5. Worth,[1] p. 56.

6. Hansell, H. F., and Reber, W.: Ocular Muscles, Philadelphia, P. Blakiston's Son & Co., 1912, p. 166.

consider it a condition in which there is marked loss of vision, without a visible defect, not amenable to treatment. A third view is based on a combination of these extremes. In this group the amblyopia may be the cause of the squint or may only be associated with the deviation of the eye.

Peter [7] and Abraham [8] and many others have attempted to classify amblyopia. No matter what conception of this disorder one possesses, one finds several types. A type found in young children which seems to be a suppression disappears completely and spontaneously as soon as glasses are prescribed. This, of course, should not be considered true amblyopia. A second type improves after atropinization and occlusion of the fixing eye. The third type is not amenable to treatment, and though definite improvement in the visual acuity cannot be determined, the patient recognizes objects better after occlusion of the fixing eye than before. Stereopsis of large objects may be present in all types. For example, if the unaided visual acuity is 20/25 in the right eye and 20/50 in the left eye, and with the appropriate correction, which is equal in the two eyes, at the time of refraction the vision is 20/20 in the right eye and 20/20 in the left eye, the condition is not true amblyopia but a temporary suppression.

On the other hand, if the visual acuity of the amblyopic eye is unimproved for several weeks and then gradually improves without occlusion and atropinization of the fellow eye to 20/40 or better, I should consider the condition suppression amblyopia. Again, if occlusion and atropinization are necessary to obtain improvement of vision, true amblyopia apparently exists.

In a third type of amblyopia the visual acuity in the amblyopic eye is 20/200 or less and rarely improves. If improvement in vision occurs, it is seldom for more than one or two lines of the Snellen chart. This is especially true of amblyopic squint associated with false projection and with poor fixation or absence of fixation. This third type of amblyopic squint is usually associated with (1) a high or low degree of deviation and (2) much variation of the angle of squint, especially if false projection is present.

STRABISMUS DUE TO OR ASSOCIATED WITH NEUROMUSCULAR DEFECTS

I am not attempting to say that defective abduction of a convergent eye or defective adduction of a diverging eye is a result or a cause of the squint. In similar manner an overacting internal rectus or oblique muscle may be the cause of or may coexist with the deviation. The

7. Peter, L. C.: Am. J. Ophth. **15**:493 (June) 1932.

8. Abraham, S.: Amblyopia: Classification, with a Report of Cases, Arch. Ophth. **12**:39 (Sept.) 1934.

important point is that these defects exist and must be partially or completely overcome to produce a satisfactory recovery from the strabismus.

The types of squint associated with muscular defects can be subdivided roughly as follows: (*a*) those associated with horizontal defects, (*b*) those associated with vertical defects and (*c*) those associated with combined horizontal and vertical defects.

A careful study of the neuromuscular coordination and the ability of a person with squint to maintain an extreme position of abduction during the objective examination is important, because, it will give an insight into the prognosis and the routine treatment in such a case. Sometimes the motility of the eyes must be determined at least six different times without cycloplegia and once under cycloplegia before a definite decision can be made that a neuromuscular defect exists. The reason for suggesting repeated examination is that defects may not be present at all times or may be overlooked during a single examination but may appear if examinations are repeated. Determination of the motility under atropinization may also disclose a defect to be spastic rather than structural. That is, overaction of an internal rectus or an inferior oblique muscle may be due to a spasm of these muscles and not to an abnormal structural shortening. This is not common.

Monocular and binocular determination of the motility are also important, because a defect may be absent binocularly, yet a marked neuromuscular defect may exist monocularly.

The differential diagnosis between spasm of a muscle and a definite defect in length is extremely important. A convergent spasm may become less or disappear when the eyes are atropinized, while an anatomic defect will not change materially.

The most simple type of defective motility is that in which no sclera is visible between the canthus and the limbus in abduction or adduction but a marked nystagmoid movement is present. In other cases the hyperadduction may be associated with a moderately high degree of accommodative convergence, which relaxes gradually under cycloplegia.

As a vertical component enters into a case, the prognosis usually becomes worse, and operative intervention may become necessary. For example, suppose a convergent squint is associated with overaction of the internal rectus and the inferior oblique muscle and also with underaction of the external rectus muscle. Such a condition affords a poorer prognosis than when underaction of the external rectus muscle exists alone.

Even though orthoptic training in cases of the muscular defect type of squint may produce parallelism of the visual axis, recurrence of the squint is the rule, because good duction power cannot always be developed.

ANISOMETROPIC STRABISMUS

Persons with anisometropia sometimes complain of an incongruity of the retinal images. Ames and Ogle [9] discussed the size and shape of the retinal images. They expressed the belief that a difference in the size and shape of the retinal images as low as 0.5 per cent may produce the asthenopic symptoms of headaches and photophobia.

Bielschowsky [10] reported a case of strabismus due to incongruity of the retinal images in which relief was obtained when the aniseikonia was corrected by proper lenses. There seems no doubt, therefore, that in some cases strabismus is associated with such abnormalities. Persons with aniseikonia and without other abnormalities, such as defects in vision, motility or fusion, are placed in this group. In such cases, when vision is good attempts should be made to correct the difference in the size of the retinal images by the method of Ames and his associates. As yet nothing has been done in this direction in the orthoptic clinics at Northwestern University and at the Children's Memorial Hospital.

Anisometropic and aniseikonic strabismus should be included in the classification for completeness. Nothing definite as yet can be said regarding aniseikonia and its association with strabismus.

COMMENT

During the last three years 354 persons with nonparalytic strabismus were admitted to the orthoptic clinic of the Children's Memorial Hospital, and 156 persons with the same type of strabismus were admitted to the orthoptic clinic of Northwestern University. Because of poor attendance and insufficient data, only 148 persons from the former clinic (group 1) and 65 from the latter (group 2) are considered to furnish suitable data for this report.

In table 3 are presented the results obtained in cases of all types of squint in group 1 by correction of the refractive errors, atropinization of one or both eyes and occlusion of the fixing eye; no fusion training is noted. These results are recorded with reference to the improvement in the angle of squint.

Thus in this group of 148 patients those with certain types of squint, such as squint associated with muscular defects and squint associated with muscular defects and amblyopia, did not recover, whereas 9 per cent of those with squint associated with fusion defects recovered and 15 per cent of those with squint associated with amblyopia recovered. Recovery occurred in 70 per cent of those with the accommodative type of stra-

9. Ames, A., Jr., and Ogle, K. N.: Size and Shape of Ocular Images: Visual Sensitivity to Differences in the Relative Size of Ocular Images of the Two Eyes, Arch. Ophth. **7**:904 (June) 1932.

10. Bielschowsky, A.: Am. J. Ophth. **18**:925 (Oct.) 1935.

bismus, and in 74 per cent of those with the accommodative-amblyopic type.

These figures also disclose the interesting fact that as the accommodative factor becomes associated with those types of squint which do not respond readily to nonsurgical treatment, the percentages of recoveries increases. Thus 20 per cent of the persons with the accommodative muscular defect type recover, whereas none of those with the pure muscular defect type recover.

TABLE 3.—*Results in a Control Series According to Type of Squint*

Types of Squint	No Improvement, No. of Cases	Improvement, No. of Cases	Recovery, No. of Cases	Total	Recovery, Percentage
Pseudoparalytic type	3	..	..	3	0
Amblyopic type	11	..	2	13	15
Accommodative type	2	7	21	30	70
Muscular defect type	12	2	0	14	0
Anisometropic type, with retinal images of different size	1	..	1	2	50
Fusion defect type	10	..	1	11	9
Combined types					
Accommodative-muscular type	16	4	5	25	20
Accommodative-amblyopic type	6	1	20	27	74
Muscular-amblyopic type	10	..	..	10	0
Muscular-accommodative-amblyopic type	5	4	1	10	10
Undiagnosed (instabilities)	2	..	..	2	0
Multiple combinations	1	..	..	1	0

TABLE 4.—*Results of Orthoptic Training Given According to the Type of Squint*

Types of Squint	No Improvement, No. of Cases	Improvement, No. of Cases	Recovery, No. of Cases	Total	Recovery, Percentage
Pseudoparalytic type	1	..	..	1	0
Amblyopic type	4	2	4	10	40
Accommodative type	1	0	5	6	83
Muscular defect type	9	2	2	13	15
Anisometropic type	0	2	2	13	50
Fusion defect type	2	0	0	2	0
Accommodative-muscular type	1	2	3	6	50
Accommodative-amblyopic type	2	0	9	11	82
Muscular amblyopic type	7	0	2	9	22
Muscular-accommodative-amblyopic type	3	0	0	3	0
Multiple combinations	1	..	..	1	0
Undiagnosed (instabilities)	0	1	0	1	0

In table 4 are presented the results of orthoptic training in cases of squint at Northwestern University (group 2). The training extended from five months to two years. Results are recorded as "no improvement," "improvement" and "recovery." There must be an absence of squint for far and near vision but not for distant vision alone before recovery is considered complete.

Even though it is usually thought that the chances for recovery decrease with increasing age, group 2 contains more subjects of advanced years than group 1. Yet, 15 per cent of those with squint associated

with muscular defects recovered and 22 per cent of those with squint associated with muscular defects and amblyopia recovered. It will be remembered that the younger patients with these two types of squint (group 1) who had no fusion training did not recover. Of those in group 2 with the amblyopic type of squint, 40 per cent recovered; in group 1, only 15 per cent recovered. Of those in group 2 with the accommodative type of squint, 83 per cent recovered, while in group 1, 70 per cent recovered.

The inevitable conclusion is: Fusion training has a definite favorable influence on most types of strabismus, even though the angle of squint does not entirely disappear.

Table 5 contains a record of the results in those cases previously classified with reference to the degree of squint. This table is important,

TABLE 5.—*Frequency of Recovery in Cases of High and Low Degree of Squint*

	Group 1 Children's Memorial Hospital				Group 2 Northwestern University			
Types of Squint	Over 15°	15° or Less	Total	Recovery	Over 15°	15° or Less	Total	Recovery
Pseudoparalytic type	3	0	3	0	1	0	1	0
Amblyopic type	10	3	13	2	3	7	10	4
Accommodative type	19	11	30	21	3	3	6	5
Muscular defect type	12	2	14	0	10	3	13	2
Anisometropic type	2	0	2	1	0	2	2	1
Fusion defect type	10	1	11	1	1	1	2	0
Accommodative-muscular type	19	6	25	5	4	2	6	3
Accommodative-amblyopic type	19	8	27	20	3	8	11	9
Muscular-amblyopic type	10	0	10	0	5	4	9	2
Muscular-accommodative-amblyopic type	9	1	10	1	2	1	3	0
Multiple combinations	1	0	1	0	0	1	1	0
Undiagnosed (instabilities)	2	0	2	0	1	0	1	0

because it discloses that most persons in groups 1 and 2 with strabismus of the pseudoparalytic, muscular defect and fusion defect types have squint of over 20 degrees. This high degree might explain why so few of these patients recover, there being none in group 1 who recovered and only 2 in group 2. But this conclusion must be incorrect, because 19 of 30 persons in group 1 with the accommodative type of squint had squint of over 15 degrees and yet 21 recovered, 6 of those being persons with over 20 degrees of squint. There was an equal number of persons with a low and high degree of squint of the accommodative type in group 2 and yet only 1 of those with over 15 degrees of squint failed to recover with fusion training. Similarly, of those with squint of the accommodative-amblyopic type in group 1, 19 of 25 had over 15 degrees of squint. It must be concluded, therefore, that in cases of strabismus associated with muscular defects recovery is poor because of a weak musculature which cannot be improved by duction training. This condition is classified as a divergence insufficiency in association with convergent strabismus. With this same classification (Duane's), in most cases the accommodative type of squint may be considered due to

convergence excess if the squint is convergent and to divergence excess if it is divergent. Hence it must be conceded that though the degree of squint has an effect on the recovery of strabismus, other factors, such as poor motility power, are equally important.

In table 6 the frequency of occurrence of each type of squint and the frequency of recovery in both groups are recorded in percentages. It can be seen that the muscular, the amblyopic and the muscular-amblyopic types of squint occur only half as frequently in group 1 (average age, 6 years) as in group 2 (average age, 10). These are the types of strabismus in which spontaneous recovery seldom occurs and in which the prognosis with nonoperative treatment is poor. This observation that the severe types of squint occur more often in older persons should stimulate those interested in orthoptic training to begin such procedures early. The fact that 40 per cent of persons with the

TABLE 6.—*Percentages of Types of Squint in Both Groups; Percentages of Recovery*

Types of Squint	Group 1		Group 2	
	Incidence	Recovery	Incidence	Recovery
Pseudoparalytic type	2.0	0	1.53	0
Amblyopic type	87.0	15	15.38	0
Accommodative type	20.0	70	9.1	83
Muscular defect type	9.39	0	20.0	15
Anisometropic type	1.28	50	3.07	50
Fusion defect type	7.43	9	3.07	0
Accommodative-muscular type	16.89	20	9.23	50
Accommodative-amblyopic type	18.24	74	16.92	82
Muscular-amblyopic type	6.75	0	13.84	22
Muscular-accommodative-amblyopic type	6.75	10	4.6	0
Multiple combination	0.6	0	1.53	0
Undiagnosed (instabilities)	1.3	0	1.53	0

amblyopic type of squint and 15 per cent of those with the muscular defect type recover by fusion training should stimulate one to attempt such training before considering surgical procedures.

The accommodative and the accommodative-muscular type of squint were almost twice as frequent in group 1 as in group 2. Here, too, fusion training is seen to be of benefit.

CONCLUSIONS

A classification of the types of nonparalytic squint is presented. This is not considered an all-inclusive classification but is offered as a convenient basis for determining the type of treatment.

Although the muscular defect, amblyopic and muscular-amblyopic types of strabismus are not benefited as much by nonsurgical treatment as the accommodative type and its combination, orthoptic training improves the condition in many cases.

The degree of squint affects the results secured by nonsurgical measures, but other factors, such as defects in motility, defects in vision and defects in fusion, may equally prevent recovery.

VITAMIN C AND ITS RELATION TO CATARACT

ESTELLE E. HAWLEY, PH.D.*
AND
OVID PEARSON, M.D.
ROCHESTER, N. Y.

The excellent experimental work and reviews which have appeared, especially in the last two years, from the department of ophthalmology of Northwestern University Medical School have increased the interest in the possible relation between the onset of cataract and the body stores of vitamin C (cevitamic acid). Delay in the development of cataract, possibly through dietary means, is a matter of such practical importance that a study of patients available in the Strong Memorial Hospital and the Rochester Municipal Hospital seemed worth while.

A brief summary of the literature may well precede a report of our laboratory findings. Bourne,[1] in her recent discussion of metabolic factors in the production of cataract, stated that "the physical and metabolic integrity of the lens is to some extent dependent upon the maintenance of a normal metabolism of the whole organism. General metabolic disturbance may affect the lens by creating a deficiency of some substance essential to lens metabolism by producing toxic substances, by altering the composition of the aqueous in respect to the water, salt or hydrogen ion concentration or by other metabolic disorders as yet unknown." Bourne considered all possible factors which might alter the normal metabolic function of the lens. We here confine ourselves to but one phase—the possible relation of cevitamic acid.

Glick and Biskind[2] stated: "The lens of the eye, having no blood supply, is especially dependent on those intracellular substances which form oxidation-reduction systems for the maintenance of many of its metabolic processes." They tabulated the concentration of cevitamic acid in the lenses of a number of animals as reported by various workers and found a range from 2 mg. per hundred grams of tissue in the rat

* Working under a grant from the California Fruit Growers Exchange.

From the Departments of Pediatrics and Ophthalmology, University of Rochester School of Medicine, and the Strong Memorial Hospital and the Rochester Municipal Hospital.

1. Bourne, M. C.: Metabolic Factors in Cataract Production, Physiol. Rev. **17**:1 (Jan.) 1937.

2. Glick, D., and Biskind, G. R.: Studies in Histochemistry: X. Distribution of Vitamin C in the Lens of the Eye, Arch. Ophth. **16**:990 (Dec.) 1936.

to 104 mg. in fish. The value for the lens in man is given as 31 mg.[3] This agrees with the work of Müller and Buschke,[4] who found the normal human lens to contain 30 mg. of cevitamic acid per hundred grams of tissue. This high percentage of vitamin C in the lens of the human eye naturally raises the question as to its origin and function. Why should vitamin C be found in much lower concentrations in the cataractous lens? Bellows[5] has reported that subjects with cataract have a lower concentration of cevitamic acid in the blood than do normal subjects, an average of 0.605 mg. per hundred cubic centimeters for the group with cataract against 1.02 mg. for the group of normal subjects. Single morning specimens of urine showed a lower excretion of vitamin C for his six patients with cataract than for six normal persons of a corresponding age, the amount excreted being expressed as milligrams of vitamin C per cubic centimeter of urine.

Glick and Biskind[2] have demonstrated that age in itself is a factor in establishing the level of cevitamic acid in lens as well as in other tissues. They found the level of vitamin C in the lens of a cow to be from 10 to 15 per cent lower than that in the lens of a calf. Such a difference has been found by various writers for all other tissues. Those tissues which have the highest metabolic activity or those taken from the growing young are always higher in vitamin content than the tissues of mature animals. The vitamin C content of mature and immature cataract,[6] however, is much too low to be accounted for by the factor of age alone.

The ability to produce cataract in the albino rat through poisoning with naphthalene,[7] or with excessive amounts of galactose, as first employed by Mitchell and Dodge,[8] has made possible studies of the effect of the administration of vitamin C on the onset of cataract. Bellows[9] was able to show that the administration of cystine and, to

3. von Euler, H., and Malmberg, M.: Neue Versuche über Askorbinsäure (C-Vitamin) in tierischen Augenlinsen, Arch. f. Augenh. **109**:225, 1935.

4. Müller, H. K., and Buschke, W.: Vitamin C in Linse, Kammerwasser und Blut bei normalem und pathologischem Linsenstoffwechsel, Arch. f. Augenh. **108**:368, 1934.

5. Bellows, J.: Biochemistry of the Lens: V. Cevitamic Acid Content of the Blood and Urine of Subjects with Senile Cataract, Arch. Ophth. **15**:78 (Jan.) 1936.

6. Bellows, J.: Biochemistry of the Lens: VII. Some Studies in Vitamin C and the Lens, Arch. Ophth. **16**:58 (July) 1936.

7. Müller, H. K.; Buschke, W.; Gurewitsch, A., and Brühl, F.: Vitamin C in Kammerwasser und Linse: Seine Bedeutung für Physiologie und Pathologie des Linsenstoffwechsels, Klin. Wchnschr. **13**:20 (Jan. 6) 1934.

8. Mitchell, H. S., and Dodge, W. M.: Cataract in Rats Fed on High Lactose Rations, J. Nutrition **9**:37 (Jan.) 1935.

9. Bellows, J.: Biochemistry of the Lens: IX. Influence of Vitamin C and Sulfhydryls on the Production of Galactose Cataract, Arch. Ophth. **16**:762 (Nov.) 1936.

a lesser extent, of vitamin C delayed the onset of the cataracts in the experimental groups. He wrote: "Galactose in some way causes a loss of the sulfhydryl content of the crystalline lens. . . . If it could be shown that they diminish in amount before the opacities in the lens appear, this, coupled with the fact that an excess of these substances in the diet delays the onset of opacities, would make it reasonably certain that the loss of these substances is responsible for cataract formation." Von Euler and Malmberg [8] found the vitamin C content of the lens of normal rabbits and guinea pigs to be 16 mg. per hundred grams, while the value for the lens of such animals rendered scorbutic by dietary restriction was diminished to 10 and 0.5 mg., respectively. The administration of large doses of vitamin C raised the level as high as 26 mg. They further reported that the value of 31 mg. for the normal human lens is diminished to 5 mg. by the presence of cataract.

A further experiment by Bellows [9] showed a diminished quantity of sulfhydryls (glutathione and cysteine) but little change in the vitamin C content before there was any visible change in the lens. Since, however, vitamin C to some extent delayed the onset of cataract, "one is led to believe that the sulfhydryls and vitamin C are partially interchangeable in carrying out oxidation and reduction in the crystalline lens. Thus, as the sulfhydryl content diminishes, vitamin C, if available, will take over more and more of this function and will keep the lens more or less viable for some time. One may theorize that senile cataract in man may arise in a similar way . . ."

The question as to whether the absence of cevitamic acid precedes the formation of cataract or is a result of it cannot yet be answered. The possibility of a low vitamin C content predisposing to the formation of cataract suggests itself.

A question arises here, as with other determinations of cevitamic acid by titration with 2,6-dichlorophenolindophenol: Is the method specific for cevitamic acid, especially when it is so closely associated with large amounts of glutathione and cysteine? Conflicting reports appear, but again one turns to the experiments of Bellows and Rosner,[10] who by the use of the enzyme from the Hubbard squash found that at p_H 2 the reduction of the dye is due to vitamin C. This confirms the experiments of Demole and Müller,[11] who found the vitamin C content, as indicated by the biologic test, to be in good agreement with that indicated by the chemical test, if loss of vitamin C during handling were

10. Bellows, J., and Rosner, L.: Biochemistry of the Lens: VIII. New Proof of the Presence of Vitamin C in the Crystalline Lens, Arch. Ophth. **16**:248 (Aug.) 1936.

11. Demole, V., and Müller, H. K.: Ueber das Vorkommen von Ascorbinsäure in Linse und Kammerwasser, Biochem. Ztschr. **281**:80, 1935.

prevented. This also further confirms the observations on the chemical method for the estimation of vitamin C by Ahmad.[12]

In the present study patients admitted to the ophthalmologic service of the Strong Memorial Hospital were followed. It is unfortunate that complete data are not available for all. The vitamin C content of the blood was determined for 43 patients; of the urine, for 48 patients, and of the lens, for 36 patients, but complete studies were made on only 16. The data are tabulated for convenience and presented in the tables, the average values for the entire group being used in all tables except table 4, which contains the complete data on 16 patients.

The values for the lens are expressed as milligrams of cevitamic acid per hundred grams of moist lens; for the blood, as milligrams per hundred cubic centimeters of whole blood, and for the urine, as total milligrams in a twenty-four hour specimen. The values for the blood were obtained on patients during fasting. No source of vitamin C was given during the collection of the "basal" specimens of urine, since it was desired to ascertain the saturation level at the time of the patients' admission to the hospital in order to obtain the full effect of the previous dietary habits on the stores of the vitamin C in the body tissues.

Methods of ascertaining the cevitamic acid content of the blood, urine and tissue are reported elsewhere.[13]

From the data presented in the tables it can be seen that in our series there is no correlation between the vitamin C content of the lens and the age of the patient or between the age or the vitamin C content of the urine and the vitamin C content of the lens, but that there is a correlation between the vitamin C content of the urine and that of the blood.

There is a possible correlation between age and the vitamin C content of the blood, as indicated in table 1. If one calculates the average age of the cataractous and normal patients studied by Bellows,[5] one finds that with the difference in the vitamin C content of the blood in the two groups there is correspondingly an age difference. The group of cataractous patients, with an average vitamin C content of the blood of 0.605 mg. per hundred cubic centimeters, have an average age of

12. Ahmad, B.: Observations on the Chemical Method for the Estimation of Vitamin C, Biochem. J. **29**:275, 1935.

13. (*a*) Hawley, E. E.; Stephens, D. J., and Anderson, G. K.: The Excretion of Vitamin C in Normal Individuals Following a Comparable Quantitative Administration in the Form of Orange Juice, Cevitamic Acid by Mouth and Cevitamic Acid Intravenously, J. Nutrition **11**:135 (Feb.) 1936. (*b*) Stephens, D. J., and Hawley, E. E.: Partition of Reduced Ascorbic Acid in the Blood, J. Biol. Chem. **115**:653 (Oct.) 1936. (*c*) Hawley, E. E.; Daggs, R. G., and Stephens, D. J.: Effect of the Administration of Acid and Alkaline Salts upon the Ascorbic Acid Content of Guinea Pig Tissues, J. Nutrition **14**:1 (July) 1937.

66, while the group of normal subjects, with an average vitamin C content of the blood of 1.02 mg. per hundred cubic centimeters, average 14 years younger.

Specimens of urine obtained under basal conditions with one exception showed a vitamin C content below normal (average, 9 mg. for a twenty-four hour specimen). Specimens of blood obtained under basal conditions with one exception showed vitamin C contents in the lower

TABLE 1.—*Average Values for Vitamin C Tabulated on the Basis of Age*

Age	Vitamin C Content of Blood, Mg. per 100 Cc.	Number of Patients	Vitamin C Content of Lens, Mg. per 100 Gm.	Number of Patients	Vitamin C Content of Urine, Mg. per 24 Hr. Specimen	Number of Patients
Under 20	1.03	10	..	..	13	15
21-30	1.12	5	13	1	12	4
31-40	1.02	1	16	2	12	2
41-50	0.94	1	11	1	7	1
51-60	0.98	7	23	9	10	5
61-70	0.91	12	21	9	12	13
71-80	0.88	5	16	11	6	6
Over 80	0.96	2	10	1	7	2

TABLE 2.—*Data Tabulated on Basis of Vitamin C Content of Urine*

Vitamin C Content of Urine, Mg. per 24 Hr. Specimen	Vitamin C Content of Blood, Mg. per 100 Cc.	Number of Patients	Vitamin C Content of Lens, Mg. per 100 Gm.	Number of Patients	Average Age	Number of Patients
Under 5	0.85	8	24	5	66	9
6-10	0.89	12	15	4	59	12
11-15	1.08	11	25	2	43	10
16-20	0.86	1	..	..	51	2
21-25	1.01	3	14	2	54	4
26 and over	0.92	1	..	..	65	1

TABLE 3.—*Data Tabulated on Basis of Vitamin C Content of Lens*

Vitamin C Content of Lens, Mg. per 100 Gm.	Average Age	Number of Patients	Average Vitamin C Content of Lens, Mg. per 100 Gm.
Under 10 mg	37.0*	1	9.4
10-15 inclusive	64.5	16	13.23
16-20	67.6	5	18.20
21-25	61.5	6	23.26
26-30	66.0	2	29.75
31-35	65.0	4	34.20
36 mg. and over	58.0	2	42.50 and 66.00†

* The patient had a traumatic cataract.
† The patient had leukemia; the vitamin C content of the blood was 5.9 mg. per hundred cubic centimeters (footnote 13b).

normal range (average, 0.97 mg. per hundred cubic centimeters). The values for the vitamin C contents of the lens were distributed as follows: 1 above normal (30 mg. per hundred grams), 2 normal, 5 from 20 to 25 per cent low, 3 from 30 to 40 per cent low and 5 from 50 to 60 per cent low.

The lack of correlation between the values for the blood, urine and lens might be explained on the basis of the duration of the vitamin deficiency. The more recent dietary would be reflected in the vitamin C content of the blood and urine, while that of the lens might be decreased owing to a prolonged inadequacy. Accurate dietary histories

TABLE 4.—*Data on Group of Sixteen Patients for Whom Records Are Complete*

Patient	Vitamin C Content of Lens, Mg. per 100 Gm.	Age	Vitamin C Content of Blood, Mg. per 100 Cc.	Vitamin C Content of Urine, Mg. per 24 Hr. Specimen	Diagnosis
M. O.	10.40	81	1.08	6	Cataract; diabetes; blind for several years
O. D.	10.50	43	0.94	7	Posterior polar cataract, complicated
L. D.	12.50	66	0.64	13	Retinitis pigmentosa; bilateral senile cataract
G. H.	12.70	68	1.10*	22*	Subcapsular cataract
H. S.	13.20	26	0.95	12	Gonococcic vaginitis; posterior polar cataract
M. C.	17.80	73	0.89	10	Hypertensive cardiac disease
F. C.	19.50	57	0.89	15	Hypertensive cardiac disease; senile mature cataract
B. G.	20.50	57	1.14	11	Bilateral cataract, cortical and immature
T. D.	22.20	39	1.02	4	Traumatic cataract
J. L.	22.20	72	0.79	5	Arteriosclerotic cardiac disease; senile mature cataract; hypertension
E. G.	23.15	59	1.14	7	Intra-ocular hemorrhage; senile mature cataract
A. L.	24.00	66	0.61	3	Glaucoma; old iritis
J. G.	25.50	76	1.00	2	Glaucoma at 69 years; cortical cataract
W. H.	29.30*	66	0.84	4	Mature senile cataract
P. H.	30.20*	66	1.35*	12	Mature senile cataract, 8 years standing
L. J.	42.50	53	1.07	11	Glaucoma; dislocated lens

* The values are within the expected normal range. We regret that to date we have not been able to obtain a normal lens for analysis but must accept the value of 30 mg. (footnotes 3 and 4).

TABLE 5.—*Correlation of Average Values for Vitamin C in the Lens with Those in the Blood and Urine and with Age*

Lens, mg. per 100 Gm.....	10-15	16-20	21-25	26-30	31-36
Average age, years.....	57 (5)*	62 (3)	62 (5)	62 (2)	53
Blood, mg. per 100 cc.....	0.94 (5)	0.97 (3)	0.91 (5)	1.10 (2)	1.07 (1)
Urine, mg. per 24 hr. specimen	12 (5)	12 (3)	4 (5)	8 (2)	11 (1)

* The numbers of patients included in the average are given in the parentheses.

TABLE 6.—*Correlation of Average Values for Vitamin C in the Urine with Those in the Blood and with Age*

Urine, mg. per 24 hr. specimen	Under 5 mg.	6-10	11-15	16-20	21-25
Age, years.....	64 (5)*	64 (4)	54 (6)	.....	68 (1)
Blood, mg. per 100 cc.....	0.85 (5)	1.01 (4)	1.01 (6)	.....	1.10 (1)

* The numbers of patients included in the average are given in the parentheses.

are difficult to obtain and not too satisfactory. As to a possible correlation between the cevitamic acid content and the cause of cataract, none is found. Analysis of the mature cataract of eight years' standing which was removed from subject P. H. and determination of the vitamin C

content of the blood indicated that both were in the normal average range. The vitamin C content of the urine was slightly lower than the average, but, as has been pointed out, the immediate preceding diet is a definite factor in determining the level. The mature cataract of E. G. was higher in vitamin content than the traumatic cataract of T. D. or the immature cataract of B. G. A larger series of patients might, of course, alter the data.

Since the average of the values for the vitamin C content of the urine was so definitely low, the question arose as to whether old age in itself with its almost universal dietary change might not, at least in part, account for the low levels.

With the permission of Dr. Eric Green and the active cooperation of Mr. George K. Anderson, studies were made on the urine of twelve patients in the Monroe County Home. Subjects were carefully selected as to age to be controls for the group of patients with ocular conditions; that is, cataractous changes could not be detected in any of the patients. The average urinary excretion of cevitamic acid for this group was 11.71 mg. in a twenty-four hour specimen, obtained under basal conditions, with extreme values of from 5.91 to 18.20 mg. As in our subjects from the hospital, the response to a daily dose of 200 mg. of vitamin C was followed. The response to the first 200 mg. of the vitamin raised the output for the group to an average of 13.07 mg. The large retention of the vitamin (intake minus urinary content) indicated a state of tissue unsaturation. A dietary survey of previous intake indicated that the ingestion of vitamin C was low. The average vitamin C content of the urine for the entire group of patients with ocular conditions (including those on whom complete data were not obtained) was 10.60 mg. in a twenty-four hour specimen, obtained under basal conditions, with extreme values of from 2 to 24 mg. The response, to the first 200 mg. of vitamin C in this group was 13 mg. The two groups, therefore, were not unlike in tissue saturation, as evidenced by initial level and response. The patients with ocular conditions were not more depleted of their stores of vitamin C than the patients from the county home. The urinary excretion in response to successive constant doses of vitamin C continued approximately the same over a period of a week for both groups. From these two sets of data it could not be seen "that in a cataractous subject much larger quantities of vitamin C are required to raise the value in the blood plasma than in a normal person" (Bellows [5]). However, the values for persons in the same age groups whose normal diet was consistently higher in vitamin C than was that of the patients at the county home might be the same as those reported by Bellows. It is hoped to obtain some patients in the same age group who have orthopedic conditions and whose dietary

intake can be controlled for a long period to ascertain whether or not the normal response to vitamin C is lower in the elderly person than it is in the younger person irrespective of ocular abnormalities.

SUMMARY

While from our data there is a definitely decreased amount of cevitamic acid in the cataractous lens, from a normal of 30 mg. to 10 mg. per hundred grams of lens tissue, and while there is a definitely lowered vitamin content in the urine, the values for the blood are in general within a low normal range.

No correlation could be found between age and the content of vitamin C in the lens, urine and blood, except the blood-urine relation, which is physiologic, and a possible age-blood correlation.

No correlation was found between the type of abnormality in the lens and the level of vitamin C.

Dietary histories in general indicate a lowered intake of foods rich in vitamin C in the old age group, probably due to several factors:

(*a*) The comparatively recent realization of the need of vitamin C and, for its adequate intake, the inclusion of citrus fruits or uncooked fruits and vegetables in the diet. The food habits of the older generation, which are so frequently continued through life, did not include these foods.

(*b*) The decreased use of fresh fruits and vegetables due to economic stress. Many of our patients are in the "low income" group.

(*c*) The voluntary decrease of acid foods or foods hard to chew or of high roughage content which so commonly occurs with advancing years.

To quote Bellows:[5] "The absence or diminution of cevitamic acid in the aqueous and in the lens of the cataractous eyes brings forth the question of the relationship of vitamin C and cataract. Is the loss of vitamin C secondary to the changes in the lens, as Bietti has suggested, or does its diminution or deficiency precede the development of opacities in the lens?" Though adequate diet cannot fail to be more effective in the preservation of normal function than one deficient in any respect and the inclusion of sufficient amounts of food rich in vitamin C advantageous, and in spite of the contrary evidence of other investigators, one is forced to conclude from the data here presented that the low content of vitamin C in the lens may be a result of cataractous changes rather than the underlying cause and that tissue saturation as measured by the vitamin C content of the urine and blood suggests that it is probably not responsible for the onset of the cataract.

Since this paper was accepted for publication it has been the privilege of one of us to read and discuss the manuscript of a paper on the use of the Evelyn photoelectric colorimeter as a means of measuring the reducing substances in urine. Dr. Evelyn's excellent discussion indicates that undoubtedly values for cevitamic acid as heretofore determined are too high. In urine certainly other reducing substances besides cevitamic acid may enter into the reaction and increase the titration value.

CAUSTIC BURNS OF THE EYE

W. B. HUBBARD, M.D.
FLINT, MICH.

There are many cases of partial or complete blindness due to burns of the eye. Cowan and Sinclair,[1] in their recent review of blindness in Pennsylvania, reported 179 cases of blindness due to burns of the eye of different types and only 26 of blindness due to detachment of the retina. Detachment of the retina is being thoroughly investigated. Much progress has also been made in the treatment of burns of the skin. These facts considered, burns of the eye are not receiving the attention they merit. For example, many physicians continue to treat such burns with test tube neutralizing substances, although this method of treating cutaneous burns has been discontinued for some time. There are many differences between cutaneous and ocular tissues, but there are also similarities. Some of the methods used in the treatment of the one may apply in the treatment of the other.

In this paper it is desired (1) to discuss briefly changes that take place when acids and alkalis injure tissues, (2) to refer to treatments of burns of the skin, (3) to report several experimental observations and (4) to suggest certain phases of treatment.

CAUSTIC ACIDS

When an eye is burned by a caustic acid in small amounts, most of the precipitation seems to be completed at once. Theoretically, free acid may remain in the interstitial spaces, deep in the eye and at the margins. The action of this acid might be compared roughly to the action of iodine under a bandage. When there is a large excess of acid, precipitation of proteinate continues longer. Sollmann [2] stated that the action of concentrated acid consists in withdrawal of water from the tissues, formation of acid albumins and softening but not dissolving of connective tissue and epithelium. Simon [3] stated that the nucleoproteins are precipitated. In regard to albumins, Hawk [4] stated

1. Cowan, A., and Sinclair, S. M.: Causes of Blindness in Pennsylvania from Medical and Social Aspects, J. A. M. A. **107**:757-760 (Sept. 5) 1936.

2. Sollmann, T.: Manual of Pharmacology, Philadelphia, W. B. Saunders Company, 1917, pp. 125, 131 and 137.

3. Simon, C. E.: A Text-Book of Physiological Chemistry, Philadelphia, Lea Brothers & Co., 1904, p. 58.

4. Hawk, P. B.: Practical Physiological Chemistry, ed. 6, Philadelphia, P. Blakiston's Son & Co., 1920, pp. 105 and 452.

that when albumin is heated acid must be added to secure its complete precipitation. Gradle [5] found that in some cases of hot acid burns of long standing in which opacities developed there was no evidence of previous dissolution of the corneal epithelium. Undissolved epithelium may account in part for the smoothness of the coagulated area. Corneal burns from unheated caustics stain with dyes, however. This indicates that even though the epithelium may not have been dissolved, the continuity has been broken and infiltration has taken place. The insolubility of the acid precipitate probably accounts for the relatively favorable outlook of burns from acid, the insoluble precipitate protecting the cornea from further injury except from acid, which may remain free until it also reacts with adjacent tissue, without burrowing, to form a precipitate.

CAUSTIC ALKALIS

When the caustic is an alkali, the damage is more severe and continues to become progressively worse for some time. In regard to the progressive action, Cross [6] stated: "Caustic soda burns give no evidence on the first day that they will look much worse on the third day than at the time of injury." Sollmann [2] said: "The area of necrosis produced by alkalies is very soft and the compounds which alkalies form are very soluble; consequently the alkalies penetrate very deeply and their action continues for several days." Simon [3] also called attention to the solubility of certain proteins in alkali solutions, particularly nucleoproteins and nucleins. The solubility of the alkaline compounds probably accounts for the unfavorable outlook following burns from alkalis.

From the foregoing discussion it would not appear favorable to attempt to change acid precipitates or coagulates to alkaline proteinates by adding alkalis. On the other hand, it would appear helpful to change soft alkaline precipitates to insoluble ones by adding acids. This agrees with the results of my experimental study.[5a]

TANNIC ACID, SILVER NITRATE AND METHYL ROSANILINE

Tannic Acid.—Tannic acid is probably the most widely used agent for treating burns. Its action on the tissues is described in textbooks of pharmacology and, since popularized by Davidson,[7] in numerous

5. Gradle, H. S.: Battery Burns, J. A. M. A. **79**:1819-1821 (Nov. 25) 1922.

5a. Hubbard, W. B.: Treatment of Caustic Burns of the Eye, Arch. Ophth. **18**:263-266 (Aug.) 1937.

6. Cross, G. H.: Ocular Injuries: Their Treatment, Arch. Ophth. **7**:357-360 (March) 1932.

7. Davidson, E. C.: Tannic Acid in the Treatment of Burns, Surg., Gynec. & Obst. **41**:202-221 (Aug.) 1925.

articles and books. Its use as an astringent in the practice of ophthalmology is described in numerous textbooks, although it is rarely advised in the treatment of burns of the eye. Graves [8] advised it in a 1 per cent solution for dropping into the eye. For burns of the eye from alkalis Cross [6] used a 20 per cent solution of glycerite of tannic acid U. S. P. locally on a cotton swab, with excellent results.

Tannic Acid and Silver Nitrate.—Bettman [9] found that cutaneous burns treated with a 5 per cent solution of tannic acid followed by a 10 per cent solution of silver nitrate had a more favorable action than tannic acid alone.

Methyl Rosaniline and Silver Nitrate.—Branch [10] recently advised treatment of burns of the skin by spraying with a 1 per cent aqueous solution of methyl rosaniline, swabbing of the area after from three to four minutes with a 10 per cent solution of silver nitrate and then after the solution began to dry respraying the entire burned area with the solution of methyl rosaniline five times at intervals of fifteen minutes. He found that the area becomes infected less often than when treated with tannic acid.

EXPERIMENTAL WORK

Along the lines mentioned, several experiments were performed on the eyes of anesthetized rabbits. It was decided (*a*) to observe the effect with the use of tannic acid alone and (*b*) to compare the results with tannic acid, tannic acid and silver nitrate, methyl rosaniline and silver nitrate and one other combination, namely, methyl rosaniline and tannic acid.

(*a*) *Effect of Treatment with Tannic Acid.*—Three drops of a caustic alkali was instilled into one eye and 3 drops of a caustic acid into another. In each eye the injured cornea became opaque immediately, and areas of conjunctiva were also precipitated. No macroscopic break was observed in the coagulated corneas, but when fluorescein was added the injured parts were seen to stain. A similar condition obtained at the end of five hours. A 5 per cent solution of tannic acid was then instilled. The injured moist conjunctival area was immediately covered with a firmer, thicker, drier, yellowish white precipitate, and appeared in a more favorable condition for healing. Clinically, the eyes were much improved the following day and continued to improve. There was no excess moisture, little discharge and no infection during the twenty days of observation. At that time the periphery of each cornea was translucent. Over each pupil there remained a small, superficial, gradually healing ulceration of the cornea.

8. Graves, B., in Berens, C.: The Eye and Its Diseases, Philadelphia, W. B. Saunders Company, 1936, p. 499.

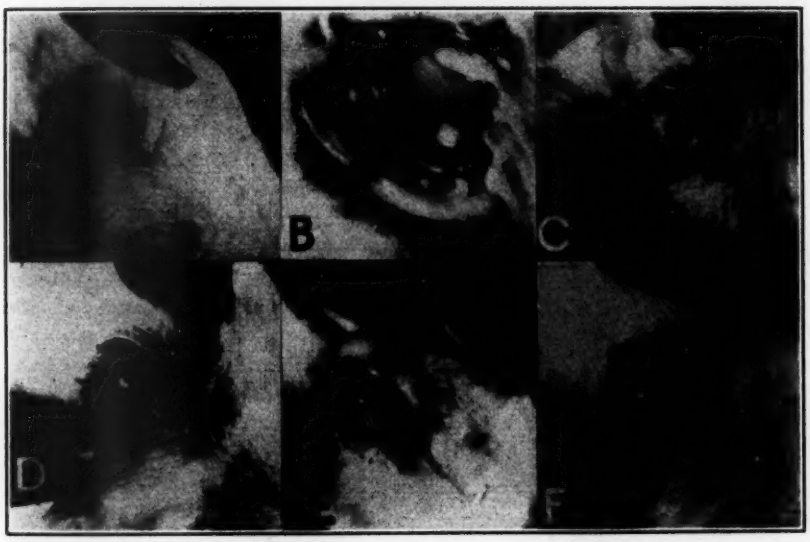
9. Bettman, A. G.: The Tannic Acid-Silver Nitrate Treatment of Burns, J. A. M. A. **108**:1490-1494 (May 1) 1937.

10. Branch, H. E.: Extensive Burns: Treatment with Silver Nitrate and Methyl Rosaniline, Arch. Surg. **35**:478-485 (Sept.) 1937.

(*b*) *Effect of Treatment with Tannic Acid, Tannic Acid and Silver Nitrate, Methyl Rosaniline and Silver Nitrate, and Methyl Rosaniline and Tannic Acid.*— The cornea of each rabbit was covered with, and the conjunctival sac was filled with, 11 drops of a 20 per cent sodium hydroxide for exactly ten seconds. The eyes were then irrigated with water. The irrigation completed the treatment of the control rabbit, but the experimental agents were at once instilled freely into the eyes of the others. Treatments with the experimental agents were repeated once the following day.

Control Experiment: After irrigation of the eye, no other treatment was given. There was early sloughing of the conjunctiva. Later rupture of the cornea occurred, with degeneration of the eye on the eighteenth day.

Tannic Acid: When a 5 per cent solution of tannic acid was instilled into the eye the cornea became stained a light yellow. The injured conjunctival elements



A, normal eye of rabbit. Note the transparent cornea, with the iris and the pupil clearly seen, and the normal lids. The nictating membrane is not as prominent as usual. *B*, rabbit's eye one day after a burn from a caustic alkali. No treatment was given. Note the opaque cornea. *C*, rabbit's eye one day after a burn from a caustic alkali. The treatment consisted of the instillation of a 5 per cent solution of tannic acid. Note the whitish gray opaque cornea. *D*, rabbit's eye one day after a burn from a caustic alkali. The treatment consisted of the instillation of a 5 per cent solution of tannic acid followed by the instillation of a 2 per cent solution of silver nitrate. Note the brilliant black stain, especially of the cornea. *E*, rabbit's eye one day after a burn from a caustic alkali. The treatment consisted of the instillation of a 1 per cent aqueous solution of methyl rosaniline followed by the instillation of a 2 per cent solution of silver nitrate and the additional use of methyl rosaniline. Note the purplish black stain, especially of the cornea. *F*, rabbit's eye one day after a burn from a caustic alkali. The treatment consisted of the instillation of a 1 per cent aqueous solution of methyl rosaniline followed by the instillation of a 5 per cent solution of tannic acid and the additional application of methyl rosaniline. Note the purplish stain, especially of the cornea.

were precipitated into a firm yellowish coagulum. However, the following day dissolution of the conjunctival coagulum was beginning, and in spite of the treatment on that day it continued. Every day the conjunctival sac was filled with flocculent or creamy discharge, and necrosis of the deeper layers of the cornea began. The lids were usually swollen shut. On the nineteenth day the eyeball had degenerated through generalized necrosis and rupture of the cornea.

Tannic Acid and Silver Nitrate: Instillation of a 5 per cent solution of tannic acid caused the changes that were described in the preceding experiment. However, when a 2 per cent solution of silver nitrate was added, the injured cornea, nictating membrane and conjunctiva were immediately stained a brilliant black. The eye seemed to progress nicely. The stain of the conjunctiva gradually decreased. There was no evidence of dissolution or infection of the coagulum. The coagulum over the cornea appeared leathery and dry. After nineteen days some sloughing of the conjunctiva and posterior soft tissues had appeared. Most of the cornea was black, although the coagulum was loosening in spots. On the twentieth day a mild orbital abscess developed. On the twenty-fifth day the abscess had subsided. The eyeball was firm and in good condition. Not until the twenty-seventh day did rupture of the necrosed cornea occur.

Methyl Rosaniline and Silver Nitrate: When a 1 per cent solution of methyl rosaniline was instilled into the eye, the membranes were immediately stained a brilliant violet. When a 2 per cent solution of silver nitrate was added the stain took on a blackish tinge. The instillation of silver nitrate was followed by the use of additional methyl rosaniline. The progress was relatively good. At the end of twenty-seven days the cornea had necrosed and ruptured.

Methyl Rosaniline and Tannic Acid: A 1 per cent solution of methyl rosaniline stained the tissues a brilliant violet. No macroscopic change was noted when a 5 per cent solution of tannic acid and additional methyl rosaniline were added. The progress in this eye after twenty-six days was similar to that in the eye treated with methyl rosaniline and silver nitrate, but somewhat better. On the twenty-ninth day the necrosed cornea ruptured.

COMMENT

The further coagulation of the soluble and partly coagulated tissue elements by agents used on them appeared favorable. Besides reacting with the residual caustic and the injured corneal tissue, apparently the tougher, denser precipitate prevented the lids from irritating the underlying parts. Likewise the injured conjunctiva appeared drier and smoother and less angry and less exposed to infection, and therefore adhesions, though seldom seen in these animals, appeared less likely to form. Contracture of the conjunctiva was not prevented. Prevention of infection appeared necessary.

The poorest result in the controlled group was obtained in the untreated rabbit.

Two rabbits treated with tannic acid showed improvement. In the controlled group the coagulum of the third rabbit, also treated with tannic acid, sloughed rapidly. This may have been due in part to an alkaline reaction of the conjunctival sac, to infection and to excess moisture from tearing. It has been noted that protein tannates are

insoluble in an acid medium while they are soluble in an alkaline medium. Antiseptics were not employed in the treatment. The eye of the third rabbit was moist.

Since a 2 per cent solution of silver nitrate produced immediately such a brilliant black reaction and such an extremely tough and tenacious coagulum, a 0.5, or not more than a 1, per cent solution would undoubtedly be sufficiently strong for the eye. However, should a tough coagulum become infected or adhere too long, one might deal with it as with similar cutaneous coagulum.

The reason for the abscess in the eye treated with tannic acid plus silver nitrate is not apparent. The black coagulum may have sealed in any residual caustic or injurious substance in a way not possible on an exposed surface of the skin. However, the condition of the cornea under the black coagulum seemed relatively good, even after the orbital abscess intervened.

Favorable results were obtained with methyl rosaniline and silver nitrate.

Excellent results were obtained with methyl rosaniline and tannic acid.

TREATMENT OF BURNS FROM ALKALIS

Emergency Treatment.—When an eye is burned with a caustic alkali it should be immediately irrigated with a weak acid or water, whichever is available. Even though some time has intervened since the burn, emergency treatment should be performed. The lids should be thoroughly opened, and the fornices reached. Irrigation should be continued until there is no possibility of free alkali remaining. If water is used for irrigation, weak acid should be applied as soon as possible thereafter. I used a 2 per cent solution of acetic acid advantageously. Weak vinegar, weak hydrochloric acid and other acids would probably be as satisfactory. An acid stronger than boric acid, however, seems desirable.

After-Treatment.—This depends somewhat on the degree of injury. A firm coagulum has been found desirable in the beginning at the site of injury. Several agents may be used, and they should reach all burned areas. Further experimental work is necessary. The following suggestions appear of value.

Instillations.—For the milder burns, frequent instillation of mild reagents seems sufficient and will not injure normal tissue. Occasional instillation of both tannic acid and another acid, such as 2 per cent solution of acetic acid, seems indicated until healing is well under way. From 1 to 2 per cent solution of tannic acid is recommended. Tannic acid jelly, 1 per cent, may be used if desired. Infection should be prevented.

In addition to asepsis, infection may be prevented in several ways, and one may use one's individual preference. A 1 per cent aqueous solution of methyl rosaniline may be instilled as an antiseptic before or after the instillation of tannic acid and may influence the coagulum favorably also. Or, as silver nitrate has been found to prevent infections and to act favorably on coagulated tissue after treatment with tannic acid, instillation from time to time of from 0.1 to 0.2 per cent solution of silver nitrate seems of value as a second choice. Excessive use of silver nitrate should be avoided, as it may result in the production of an extremely tenacious coagulum.

As more than one type of treatment has been found useful, the highly recommended tannic acid may be omitted entirely and another method utilized. For example, a 1 per cent aqueous solution of methyl rosaniline may be instilled before and after the aforementioned weak solutions of silver nitrate.

Localized Applications.—In order to secure more rapid and thorough action in the more severe burns, the agents may be used somewhat stronger in the beginning and applied locally to the injured areas, care being taken to prevent irritation of uninjured membranes.

Burns from lime may be treated by continuous heat-removing irrigation, removal of the lime and the aforementioned methods. At the earliest favorable time thereafter, vigorous treatment with neutral ammonium tartrate should be instituted.[11]

In the treatment of burns of the eye one should be reminded that burns of the skin do better when dry, i. e., in the absence of aqueous solutions, grease and oil.[7] The relatively dry eyes in the experiments also progressed more favorably. Therefore, although excessive dryness may be undesirable, it is suggested that excess moisture in the eye be avoided, especially during early after-treatment.

General local measures, such as the use of atropine, should be employed according to the indications.

TREATMENT OF BURNS FROM ACID

Emergency Treatment.—The emergency treatment of burns from acids is similar to that of burns from alkalis with the exception that water alone is used in irrigation. Any neutralization with an alkali is to be avoided.

After-Treatment.—As acid does not burrow as alkalis do, the after-treatment of the milder burns need not be so energetic. However, for severe burns, it appears in order to use the same after-treatment as is

11. Barkan, O., and Barkan, H.: Treatment of Lime Burns of the Eye, J. A. M. A. **83**:1567-1569 (Nov. 15) 1924.

used for burns from alkalis should the coagulum appear unhealthy, and to prevent infection.

SUMMARY

For emergency treatment, water and weak acids should be used freely. The use of an alkaline neutralizing fluid should be avoided.

For after-treatment, the use of alkalis should be avoided, especially in the early stages of treatment. Weak acids are of value. Treatment with tannic acid is usually to be preferred. When tannic acid is used, antiseptics should be used in conjunction with it. Methyl rosaniline and silver nitrate answer this purpose.

To those not desiring to use tannic acid, a combination of methyl rosaniline and silver nitrate is recommended.

Agents, such as atropine and compresses, should be used according to the indications.

Clinical Notes

CLINICAL EXPERIMENTS WITH ONE PER CENT SOLUTION OF EPINEPHRINE HYDROCHLORIDE

LOUIS H. SCHWARTZ, M.D., NEW YORK

Solution of epinephrine hydrochloride in a concentration of 1 : 1,000 has been used as a vasoconstrictor for the past forty years. In the practice of ophthalmology this preparation is of more or less therapeutic value for its vasoconstrictive action in cases of scleritis, episcleritis, various forms of conjunctivitis, keratitis (including ulcer), iritis, iridocyclitis, prolapse of the iris, glaucoma and pannus and occasionally in cases of a few other ocular conditions. Recently the 1 per cent strength was introduced for oral administration in cases of asthma, and about two and one-half years ago I began some clinical experimental studies with it in the field of ophthalmology, using two different preparations.[1] The two preparations acted equally well, and so far as these experiments were concerned there did not seem to be any difference in the results.

The first phase of the problem concerning the 1 per cent solution consisted in determining whether it was sufficiently safe for general use in the treatment of those same ophthalmologic conditions for which the 0.1 per cent strength was hitherto employed. Even the use of the latter, apparently, was not wholly free from danger, for in this connection Darier[2] stated: "In ophthalmology, we employ, as a rule, a 1-1000 solution of adrenalin hydrochlorid, a single drop of which is sufficient to produce a very marked therapeutic effect. It is indeed so pronounced that if it is indicated to use the remedy several times a day, weaker solutions, for example 1-5000, must be employed, since large doses of adrenalin may occasion serious complications."

Bearing this in mind, I performed the earliest experiments on the eyes of cats, rabbits and guinea pigs. At first 1 minim (0.06 cc.) of the solution of epinephrine hydrochloride a day was instilled into the animal's eye, the other eye serving as a control. The only effect noted was the marked temporary blanching of the superficial conjunctival vessels and dilatation of the pupil, which came on within a few minutes and lasted for variable periods up to about two hours. The deeper (scleral) vessels did not become ischemic. There seemed to be no systemic

Read before the Clinical Society of the Harlem Eye and Ear Hospital, Jan. 21, 1938.

1. One of these preparations was donated by Park, Davis & Company and the other by the Wilson Laboratories.

2. Darier, A.: Ocular Therapeutics, Philadelphia, P. Blakiston's Son & Co., 1910, p. 165.

effects. Next the preparation was used on patients who were absolutely blind and then on those who were virtually sightless. None of these persons experienced any discomfort, either locally (except for a stinging sensation lasting about half a minute) or systemically. After several months of observation on approximately fifty patients, it was definitely decided that there was no danger or ill effect, either local or general, from its use. In not a single instance was there any systemic reaction, and in no case did the ocular condition become aggravated. Since then I have tried the 1 per cent solution locally in the eyes of over 200 patients without a single disturbing reaction. All of them had the preparation for use at home, employing from 1 to 3 minims (0.06 to 0.18 cc.) in each eye daily, in some instances for as much as six months continuously. Most of the patients were seen once a week.

After the harmlessness of using the preparation in this manner was definitely ascertained, it was tried on patients with a large variety of ophthalmologic conditions, particularly those in which marked vasoconstriction might be an important factor in treatment. All told, I have used the 1 per cent solution of epinephrine hydrochloride in the treatment of more than a dozen diseases, including blepharitis, conjunctivitis (acute and chronic), vernal catarrh, granular conjunctivitis, trachoma, keratitis, corneal ulcer, pannus, iritis, iridocyclitis, glaucoma (acute and chronic), dacryocystitis (acute and chronic), cataract, choroiditis and retinitis. In patients suffering from various types of conjunctivitis the use of the 1 per cent solution induced a marked ischemia of the smaller superficial blood vessels. The more marked the hyperemia, the more marked the ensuing pallor. In those with simple conjunctivitis and vernal catarrh the daily instillation of this solution caused an abatement of the symptoms and seemed to clear up the redness, lacrimation and itching more rapidly than other measures in general use. Recurrences were not prevented but were readily checked whenever they occurred.

When the 1 per cent solution of epinephrine hydrochloride was employed in cases of scleritis and episcleritis, the sclera became a pearly, bluish white. The deeper vessels sometimes stood out more prominently as a result of the pallor induced in the more superficial vessels. When no other measures were used, these effects would disappear in a comparatively short time (a matter of an hour or two). However, if shortly after instillation the eye was massaged with a little yellow mercuric oxide ointment for a minute or two, the episcleral congestion cleared up more readily than otherwise. It would seem, therefore, that the 1 per cent solution of epinephrine hydrochloride is a valuable adjuvant in the treatment of scleral conditions.

In cases of keratitis the cloudiness of the cornea did not seem to be affected in any way by the 1 per cent solution, even though it was continued for several months. Nebulas and leucomas remained unchanged. Phlyctenules sometimes seemed to disappear somewhat more rapidly than otherwise. Ulcers of the cornea were not favorably affected so far as shortening or checking the process was concerned. This was to be expected, in view of the fact that the vasoconstriction retarded the healing process. However, when the solution was used for a day or two in the painful stage of the disease, some patients

obtained relief. It has been claimed that a solution of epinephrine hydrochloride has a beneficial action on pannus. I tried the 1 per cent preparation in several cases in which the condition was well marked and followed up the course by means of slit lamp examinations, but I was unable to note any appreciable change for the better. The same may be said for the use of the solution in cases of cataract, retinitis and choroiditis. When the condition was well advanced there was no improvement, even though some of the patients kept up the treatment and were under observation up to nearly two years.

A very favorable action of the 1 per cent solution of epinephrine hydrochloride was noted in persons with iritis and iridocyclitis, particularly in those who had adhesions from previous attacks and in those with a tendency to elevation of the tension. I tried the preparation on patients with iridocyclitis both with and without the use of atropine. When the solution was used alone, mydriasis did not result in all cases. When there was dilatation of the pupil, it came on within from five to ten minutes as a rule and reached its maximum in about a half an hour. It disappeared gradually within from one to two hours. When the solution was combined with atropine, it seemed to intensify the breaking up of synechiae and to shorten the course of the disease. In several cases in which the tension increased after the use of atropine, I discontinued the atropine and had the patients continue to use the solution of epinephrine hydrochloride without discomfort. Likewise, to break up adhesions, I found it advantageous to alternate the atropine and the solution of epinephrine hydrochloride a number of times. Fuchs stated that epinephrine promotes absorption of the atropine.

In cases of glaucoma, especially acute exacerbations of the chronic variety, the 1 per cent solution of epinephrine hydrochloride usually, though not always, brought about a marked and almost immediate reduction in tension. Sometimes the drop was as much as 20 mm. of mercury as measured by the Schiötz tonometer. The lowering of the tension, however, was not sustained as a rule, unless pilocarpine or physostigmine was used in conjunction with the epinephrine. Unless these preparations were combined, the action was transitory, even though several drops were instilled every two hours. Nevertheless, there was often advantage in a prompt relief from pain. As previously stated, in some cases of glaucoma no appreciable reduction in tension was obtained, even when the solution was employed regularly every four hours for a number of days.

The most dramatic action of the solution was obtained in cases of chronic suppurative dacryocystitis in which there was an acute flare-up. Within a day or two after the introduction of the solution there was a gradual but definite diminution of the inflammation, swelling and pain. This continued until the entire process had subsided, which took about one week. In several cases of this type the condition cleared up without operation. In cases of ordinary epiphora, however, no improvement occurred with this therapy.

Another condition in which the 1 per cent solution holds promise of favorable results is progressive myopia, especially in young children. In some instances the increasing myopia appeared to be arrested. It is still too early, however, to state whether the results are permanent. Nor is it possible as yet to say how long the medication must be kept

up after the myopia has become stationary. On the other hand, there were a number of cases in which there was no improvement.

Among the patients under observation were 2 with congenital nystagmus. These patients were using the 1 per cent solution of epinephrine hydrochloride for other conditions, but after a number of months it was noted several times that the nystagmus decreased. Whether this was a pure coincidence or not, I am not prepared to state. Unfortunately both patients were lost from observation, one after four months and the other after seven months.

REPORT OF CASES

CASE 1.—M. W., a housewife, aged 40, was seen on Dec. 1, 1936, with the complaint that nine weeks previously she had lost the sight of the left eye. Since then the eye had been a little red and moderately painful. According to her history, vision in the left eye had been poor for many years, during which time the eye frequently became inflamed and painful. Vision on the date of admission to the hospital was 20/20—3 in the right eye, perception of light in the left eye. Examination of the left eye showed an irregular pupil with numerous deposits on Descemet's membrane and on the anterior capsule of the lens. The anterior chamber was normal, and there was no increase of intraocular tension. The iris was discolored, and the markings were indistinct. The details of the fundus could not be visualized because of opacities of the vitreous. There were numerous synechiae which could not be broken away completely with atropine. The accessory nasal sinuses were clear. There were numerous decayed teeth, some of which were loose. The patient said she had had repeated attacks of pains in the joints.

A diagnosis of iridocyclitis was made.

The teeth were treated by a dental surgeon; several had to be extracted, and the rest were cleaned up generally. Large doses of salicylates were prescribed. Locally, atropine, hot applications and a patch over the eye were employed. The eye gradually became less painful and less inflamed, but the pupil was still irregular because of adhesions. For the purpose of breaking up the synechiae, I employed the 1 per cent solution of epinephrine hydrochloride, using 1 minim every four hours for five days. It had little effect. The tension remained normal throughout. By this time the pupil had begun to get smaller, and I employed both the solution and the atropine. Most of the adhesions broke away after four or five days. The eye gradually became white again.

In this case it is difficult to determine whether the solution of epinephrine hydrochloride was instrumental in breaking up the adhesions, as I have found that the same type of result has been obtained without it in other cases.

CASE 2.—Mrs. R. De A., a housewife, aged 48, was seen on March 14, 1936, with an acute exacerbation of chronic dacryocystitis. The history was as follows: For several years the patient suffered from excessive tearing. About six months prior to the onset of the present condition, there was pus on pressure at the inner canthus of the affected eye. The pain, swelling and inflammation developed a week before the patient presented herself for treatment.

Physical examination disclosed a brawny, tender swelling. No fluctuation was present. Hot antiseptic applications and astringents were employed for another five or six days. The mass softened, and incision and drainage were advised, but the patient refused surgical intervention. I then began using the 1 per cent solution of epinephrine hydrochloride, 1 minim every four hours. There was an immediate

response. Within two days the swelling and inflammation subsided considerably, and at the end of another week they were gone completely.

I have since used the solution in similar cases with equally good results.

CASE 3.—M. B., a housewife, aged 57, was first seen Jan. 25, 1936, with the complaint of gradual impairment of eyesight for the past three or four years. During all that time she had no pain, but about one week before the present examination she began to have headaches, mainly over the right eye. Vision was 20/200 in the right eye and 20/70—1 in the left eye. The pupils were moderately dilated, the right being slightly larger than the left. The right cornea was "steamy." The anterior chamber was shallow. The lens, fundus and media were clear. There was moderate cupping of the disk. Tension in the right eye was 70 mm. of mercury (Schiötz) and in the left eye 42 mm. In the course of one week, with the use of a 2 per cent solution of pilocarpine hydrochloride, the tension in the right eye dropped to 40 mm., and the pain subsided. On June 9, the patient was seen again because of another atack, despite the fact that she had been using the pilocarpine regularly (at least, so she said). This time the tension in the right eye was 76 mm. of mercury (Schiötz). For several days the pressure remained elevated in spite of the use of pilocarpine. On June 13 it was still 65 mm. I then used the 1 per cent solution of epinephrine hydrochloride, 1 minim every hour. After two hours the pressure dropped to 48 mm. I had the patient continue the use of the solution of epinephrine hydrochloride at home every four hours. When she returned after two days the tension was down to 40 mm., but in a couple of days it was again up to 50 mm. I then had her use physostigmine, together with the solution of epinephrine hydrochloride and the tension dropped and hovered between 38 and 40 mm. for the next few weeks.

In this case, as in others of this type, I found that the 1 per cent solution of epinephrine hydrochloride brought the tension down rather quickly but that it did not keep it down unless it was employed in conjunction with other miotics, such as pilocarpine and physostigmine.

CASE 4.—L. S., a schoolgirl aged 14 years, was seen on Oct. 2, 1931, for refraction. She had been wearing glasses for the past two and a half years. Vision was 10/200 in each eye. Refraction under atropine disclosed bilateral myopia of 5 diopters. The lenses she was wearing at the time were —4.00 D., which gave her vision of 20/30 in each eye. With a full correction, vision was 20/20. I examined the girl at least once every year since her initial visit. Her vision progressively grew worse, and she required a greater minus correction to bring it to 20/20. There was no pathologic condition in the fundi. On Dec. 26, 1936, the vision in each eye was 3/200, with a —8.00 D. lens for the right eye and a —8.25 D. lens for the left eye, vision was 20/20—. I then had her use the 1 per cent solution of epinephrine hydrochloride. For the first three months 1 minim was instilled into each eye every night. Then the dose was increased to 1 minim three times a day. During each month the solution was omitted for a period of one week. The acuity of vision with the correction gradually improved, so that on Nov. 1, 1937, it was 20/15—2 in each eye. Without correction, the vision was 3/200, as before.

I have used the 1 per cent solution of epinephrine hydrochloride in about twenty-five cases of this type. In the vast majority, the acuity of vision with full correction improved. In a few instances I succeeded

in satisfactorily reducing the strength of the minus correction. For the time being at least, the progress of the myopia has been checked in some of these cases. Further observation over a period of several years will be required before it is possible to evaluate more definitely the use of this preparation in the treatment of progressive myopia.

COMMENT

While the foregoing experimental studies were in progress, other workers have reported their experiences with the concentrated solution of epinephrine hydrochloride. Thus Barkan and Maisler [3] reported its successful use in the treatment of glaucoma secondary to uveitis. Previously Green [4] and Gradle [5] obtained highly satisfactory results in cases of glaucoma. My own results in these conditions have not been as gratifying, but the matter deserves further study.

3. Barkan, O., and Maisler, S.: Adrenalin Chloride 1:100 in Ophthalmology, Am. J. Ophth. **20**:504 (May) 1937.

4. Green, J.: Two Per Cent Epinephrine Solutions as Substitutes for Laevo-Glaucosan, Arch. Ophth. **5**:350 (March) 1931.

5. Gradle, H. S.: Use of Epinephrin in Ocular Hypertension, J. A. M. A. **84**:675 (Feb. 28) 1925.

AN IMPROVED TRIAL FRAME AND TEMPLES

NATHAN SCHNECK, M.D., MANITOWOC, WIS.

In the trial frame presented here the old time bridge is replaced by a nose grip similar to that on modern spectacle frames. The advantages of this type of bridge over the other are as follows:

1. It grips the nose on each side, thereby distributing its weight equally on the two sides rather than letting it all rest on the dorsum of the nose.

2. Once the bridge is adjusted, it cannot slip or slide.

In the case of a straight, nearly vertical nose or a deep saddle nose, the old type of bridge could hardly be kept in position unless the frame was held tightly behind the ears and at the root of the nose, which usually led to pain, so that the frame would have to be released. The new grip eliminates this difficulty. It is more convenient to put on the patient's face and it is more easily worn. Particularly is this problem of maintaining the trial frame in position encountered when children are being examined. With the new type of grip the frame rests on the nose of the child as securely as on that of the adult.

The temples on the trial frames that I have seen and used have all been too long to fit children, and even adults whose faces and heads were small. The temple shown here can be contracted enough to fit a child as comfortably and snugly as it fits an adult.

I may add that I have used a frame with this type of nose piece for the last two years exclusively and with such success that I should not want to go back to the older kind of trial frame.

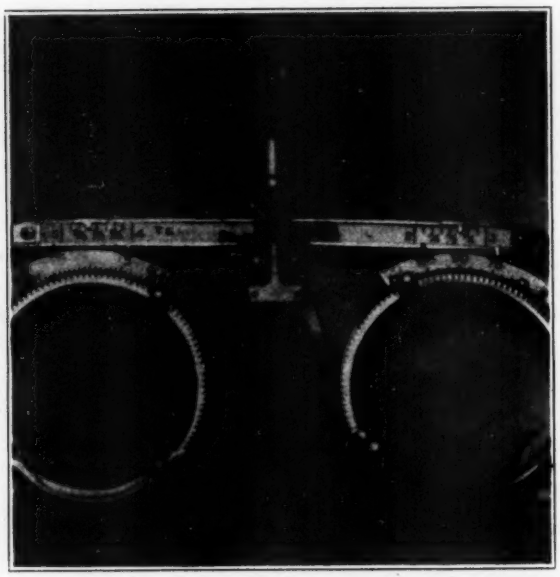


Fig. 1.—Improved trial frame. *A* indicates the knob controlling the spread of the nose pads; *B*, the screw controlling the vertical position of the nose pads, and *C*, the handle controlling their anteroposterior position.

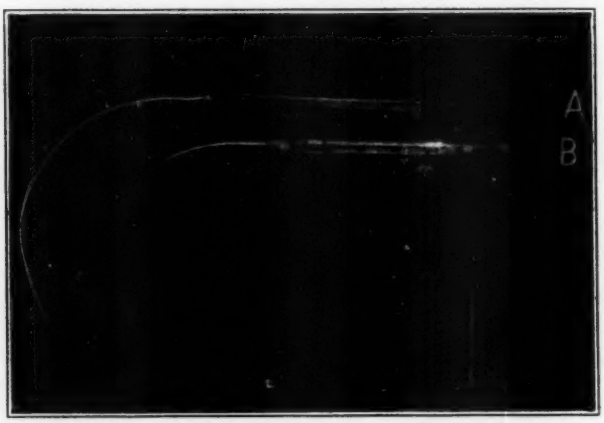


Fig. 2.—Old and new type of temple. *A* shows the shortest length of the old type of temple, and *B*, the shortest length of the new type, which can be extended longer than the old model.

A NEW MODEL GONIOSCOPE

JOHN M. McLEAN, M.D., AND ALBERT GOEBEL, BALTIMORE

This report contains a brief description of an instrument for examining the anterior iridocorneal angle. The instrument combines the advantages of several of the methods now in use and attempts to obviate some of their less desirable features.

Troncoso's [1] gonioscope has two outstanding advantages: It is conveniently portable, and it is so constructed that the entire angle may be readily examined. It gives, however, only relatively low magnification and has the definite disadvantages inherent in monocular visualization of a three-dimensional structure. Koeppe's [2] method of studying the angle gives the examiner binocular vision, but it has been generally discarded because it is rather awkward and restricts the examination to limited portions of the angle. Barkan and his co-workers [3] have described a method for mounting the corneal microscope and slit lamp so that a satisfactory binocular view of most of the angle is obtained. While this arrangement is a decided improvement, it seems a bit cumbersome, and by virtue of permanent suspension from the ceiling, the instrument is not portable. Both Bruce [4] and Castroviejo [5] have advocated use of the corneal microscope held in the examiner's hand. This method is satisfactory for a general survey of the angle. As Bruce suggests, it is useful in searching for foreign bodies, but the microscope lacks the firm, steady support necessary for prolonged study or for the use of the higher magnifications.

It seemed, therefore, desirable to construct a gonioscope with the following features: binocular vision, different magnifications, simple and convenient exploration of the angle in its entire circumference and sufficient steadiness for a careful detailed examination, yet sufficient portability to be transferred from the office to the dispensary, ward or operating room. To this end, the following instrument was constructed (fig. 1).

A stand of heavy pipe stock is mounted on a wide four-legged base (*a*) with swivel casters. The upper part of the stand is counterweighted and can be raised or lowered to any desired position (*b*). From this portion a horizontal arm (*c*), composed of two parallel members mounted on a pivoted brake drum, carries the examining and illuminating parts. The binocular head of the microscope (*d*), which may be taken from a slit lamp, is suspended, together with its rack and pinion for focusing, under the end of this horizontal arm. The complete head is mounted on an axle (*e*), so that it may be set at the various necessary angles.

From the Wilmer Ophthalmological Institute of the Johns Hopkins Hospital and University.

1. Troncoso, M. U.: Am. J. Ophth. **8**:433, 1925.
2. Koeppe, L.: Ztschr. f. Augenh. **40**:138, 1918.
3. Barkan, O.; Boyle, S. F., and Maisler, S.: Am. J. Ophth. **19**:209, 1936.
4. Bruce, G. M.: Visualization of Foreign Bodies in the Iridocorneal Angle, Arch. Ophth. **10**:615 (Nov.) 1933.
5. Castroviejo, R.: Am. J. Ophth. **19**:786, 1936.

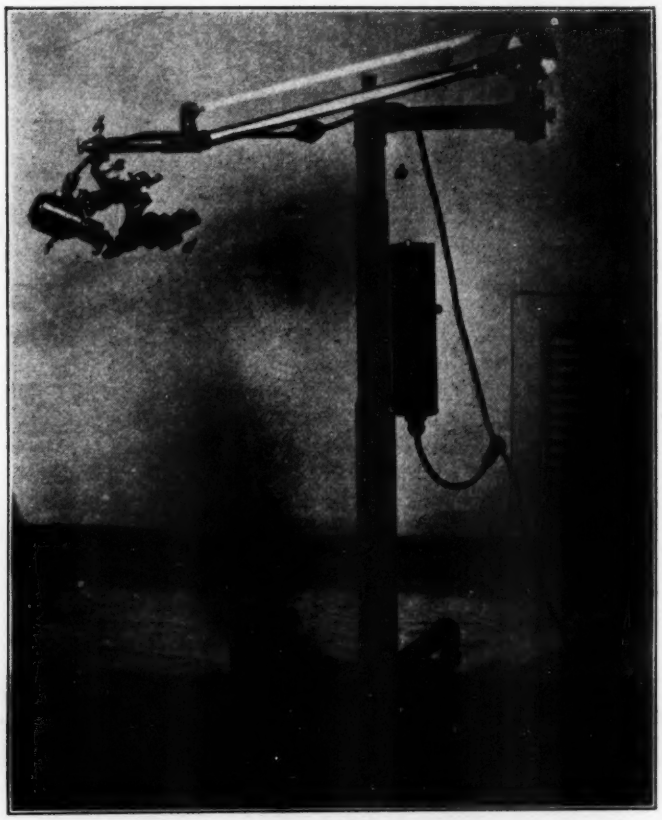


Fig. 1.—A new model gonioscope.

Fig. 2.—The patient under the instrument with the gonioscopic glass in place.

Illumination is supplied by a "hammer" lamp (*f*) mounted on an extension of the base of the microscope, so that they both move together and maintain the same relative positions. A mirror (*g*) on a universal joint and adjustable sleeve reflects the light into the eye. The entire illuminating and viewing unit is suspended by a pivot (*h*), which allows free rotation but can be locked by a set screw.

The complete instrument may be wheeled from place to place but maintains a rigid stability. Its various adjustments allow the head to be raised or lowered and swung over the patient's eye in almost any location. Examination is always conducted with the patient lying down. The usual gonioscopic glass (fig. 2) is inserted after anesthetization with pontocaine hydrochloride, and the glass is filled with physiologic solution of sodium chloride. The pivot (*h*) is centered over the eye, so that once the light and microscope are brought into focus little, if any, readjustment is necessary for examination of the entire angle, as the instrument is rotated about this point. The inverted position of the microscope and light keeps them out of range of the nose, the brow and other obstructions. Adjustment of the angle of illumination and steady binocular magnification of variable degree give a satisfactory detailed view of the angle of the anterior chamber with good depth perception.

Ophthalmologic Review

EDITED BY DR. FRANCIS HEED ADLER

PATHOGENESIS OF CHRONIC SIMPLE GLAUCOMA

A NEW CONCEPT OF THE MAINTENANCE OF THE NORMAL INTRAOCULAR PRESSURE

HERMAN ELWYN, M.D.

NEW YORK

In the literature on the mechanism of glaucoma one finds that all the theories proposed have as their basis of explanation a disturbance or change in some particular structure of the eye. These theories actually fail to explain the mechanism of chronic simple glaucoma. In the observation of patients with this disease the impression is inescapable that in its clinical aspects chronic simple glaucoma presents striking analogies to such diseases as essential arterial hypertension and diabetes. The common denominator of these diseases is, as I shall show later, a disturbance in the regulation of physiologic function. On the basis of these analogies it seemed logical to attempt to explain the mechanism of chronic simple glaucoma as primarily a disturbance in the regulation of the normal intraocular pressure. It is the purpose of this communication to present the new concept. In the following discussion I shall, first, review briefly the theories of glaucoma; second, discuss the prevailing understanding of the normal intraocular pressure and show the inadequacy of the theory of a hydrostatic-osmotic equilibrium; third, present my own conception of the maintenance of the normal intraocular pressure, and, fourth, present a new concept of the pathogenesis of chronic simple glaucoma.

Although much of what I have to say is theoretic and conjectural, these theories, I believe, present a logical sequence which helps in the understanding of this disease.

THEORIES OF THE MECHANISM OF GLAUCOMA

The theories which attempt to explain the mechanism of glaucoma have been reviewed within recent years by Gifford,[1] Thiel [2] and Peters,[3]

1. Gifford, S. R.: The Pathogenesis of Glaucoma, Arch. Ophth. 3:88 (Jan.) 1930.

2. Thiel, R.: Die Physiologie und Pathologie des Augendruckes; Glaukom, in Schick, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1931, vol. 4, pp. 666 and 700.

3. Peters, A.: Das Glaukom, ed. 3, Berlin, Julius Springer, 1930.

and their publications contain extensive bibliographies. For my purpose I shall briefly review the important theories.

Since the intraocular pressure is dependent on the tension exerted on the scleral coat by the intraocular structures, most of these structures have been made the basis on which theories of mechanism of glaucoma have been built. They include the sclera, the iridocorneal angle, the ciliary body, the vitreous and the choroid. These theories may be classified as follows: (1) retention theories, based on a hindrance to the elimination of the aqueous; (2) a theory based on an increase in the production of aqueous, and (3) a theory based on an increase in the volume of one or more of the intraocular structures.

RETENTION THEORIES

Retention Due to a Change in the Sclera.—According to this theory, the sclera loses its elasticity and shrinks, and by its shrinkage hinders the proper outflow of the aqueous. The loss of elasticity is due either to a senile change or to the sequence of inflammatory changes.

Retention Due to Blocking of the Drainage of the Aqueous at the Iridocorneal Angle.—Blocking of the angle followed by an increase in intraocular pressure has been produced experimentally and is frequently the cause of secondary glaucoma. In cases of chronic simple glaucoma blocking has been assumed to be due (*a*) to pressure of the iris against the angle, the iris being pushed forward either by a large lens or by a hypertrophied ciliary body or the whole lens-iris diaphragm being pushed forward from behind; (*b*) to inflammatory changes in the anterior segment of the eye followed by adhesions between the iris and the cornea; (*c*) to the deposits of pigment in the pectinate ligament; (*d*) to primary sclerosis of the pectinate ligament; (*e*) to changes in the endothelium lining Schlemm's canal; (*f*) to an inability of the angle to open widely, and (*g*) to a combination of several of these factors.

In objecting to these theories based on the blocking of the iridocorneal angle it may be said that all the changes found in the angle in cases of chronic simple glaucoma can be viewed as the consequence of glaucomatous tension. The angle is frequently found to be open and is always open in the early stages. Troncoso,[4] who recently reviewed the subject, examined 34 eyes with simple glaucoma and found the angle open in 19, partly closed in 13 and closed in 2. He concluded that in cases of simple glaucoma peripheral synechia is the result and not the cause of the increase in pressure. Inflammatory changes have not been found in the angle in the early stages of simple glaucoma, and

4. Troncoso, M. U.: Closure of the Angle of the Anterior Chamber in Glaucoma, Arch. Ophth. **14**:557 (Oct.) 1935.

the deposit of pigment is a senile change frequently found in the iridocorneal angle in nonglaucomatous eyes. Pigment in the angle is more frequently found in the Japanese than in members of the white race, although glaucoma is not more frequent in Japan.

Retention Due to Blocking of the Drainage of the Aqueous in the Iris as a Result of a Deposit of Pigment or of Degenerative Changes.—A deposit of pigment in the iris has the same significance as that in the angle, and degenerative changes may well be considered as secondary to the increase in tension.

THEORY BASED ON AN INCREASE IN THE PRODUCTION OF AQUEOUS

An increase in the production of aqueous has been assumed to be due to disturbances in innervation and to inflammatory and degenerative causes. Aside from the fact that no such changes have been found in the early stages of glaucoma, such a theory does not explain why the removal does not keep pace with the increased production.

THEORY BASED ON AN INCREASE IN THE VOLUME OF THE INTRAOCULAR STRUCTURES

The Vitreous.—The vitreous, behaving as a colloid, swells when brought to an acid reaction. It has been assumed that there occurs in glaucoma a change in the reaction of the vitreous toward the acid side, causing it to swell and thus increasing the intraocular pressure. Investigation has shown that normally the p_H of the vitreous is around 7.5 to 7.6, and any change in the reaction toward the acid side causes a diminution in the volume of the vitreous until the isoelectric point, which is 4.2, is reached.

The contrary has been assumed by others, that is, that the vitreous becomes more alkaline and swells. Critical investigation seems to show that the vitreous in the living eye is at its maximum turgescence.

Other theories based on an increase of the fluid content of the vitreous or on the difficulty of fluid leaving the vitreous seem to have little in support.

The Choroid.—The choroid is a highly vascular membrane which can easily change its thickness by varying its blood content. A number of theories as to the cause of glaucoma have been built on this fact, as follows:

(*a*) Obstruction of the vortex veins occurs owing to kinking of the veins at the sinus or to endophlebitic or sclerotic processes in the veins. The last factor has been especially emphasized by Magitot and

Bailliart [5] and has been fortified by the fact that when the vortex veins are tied experimentally the intraocular pressure quickly rises. Friedenwald,[6] who repeated Koster's experiments of ligating the vortex veins, found that the increase in pressure and the other changes were only transitory and that in a short time the pressure returned to normal.

(*b*) A relaxation of the vasomotor mechanism causes a relaxation of the uveal vessels and an increase in the blood content of the eye. This is the view of Hamburger,[7] who bases his treatment of glaucoma with epinephrine and epinephrine substitutes on it.

An increased tonicity of the general sympathetic nervous system as well as a disturbance in the function of the sympathetic nerve fibers in the eye has been assumed by others to be the cause of glaucoma. While relaxation or dysfunction of the sympathetic nervous system may be a factor in acute congestive glaucoma, there does not seem to be any proof that it plays any role in the causation of chronic simple glaucoma.

Thiel,[2] in reviewing the theories concerning the mechanism of glaucoma, came to the conclusion that there are a number of local and general factors which play a role in the etiology of glaucoma, including disturbances in the regulation of the intraocular vascular apparatus which cause changes in the circulation and in the exchange of fluid between blood and tissues. Since the vessels are under the control of the sympathetic nervous system, there is probably a close relation to the endocrine and the vegetative nervous systems. Thiel concluded that essentially the pathogenesis of primary glaucoma is still unknown.

SUMMARY

On reviewing all these theories, I find that each and all attempt to explain the production of glaucoma on the basis of a disturbance or change in some individual structure of the eye or in a combination of several of them. A close study of these theories brings the conviction that they are insufficient to explain the pathogenesis of glaucoma. Some authors, like Thiel, hint at a disturbance in regulation, at a multiplicity of factors and at a relation to the endocrine and sympathetic nervous systems in a vague sort of way. However, no definite theory which is based on a disturbance in regulation has so far been advanced.

5. Magitot, A., and Bailliart, P.: The Circulatory Regime of Glaucoma, Am. J. Ophth. **8**:761, 1925.

6. Friedenwald, J. S.: The Pathogenesis of Acute Glaucoma, Arch. Ophth. **3**:560 (May) 1930.

7. Hamburger, C.: Ueber die Ernährung des Auges, Leipzig, G. Thieme, 1914.

NORMAL INTRAOCULAR PRESSURE

A complete review of the physiology of the intraocular fluids and of the normal intraocular pressure is given by Duke-Elder [8] in the first volume of his "Textbook of Ophthalmology" and by Adler.[9]

The normal intraocular pressure ranges from about 18 to 25 mm. of mercury. During the twenty-four hours of the day there are slight variations of about 2 or 3 mm.; the pressure is highest in the early morning and lowest in the late afternoon. In the normal person with no tendency to glaucoma the normal intraocular pressure persists throughout life. How is this pressure maintained?

The eye is a globe made up of a slightly distensible coat and containing solid, semisolid and liquid structures which occupy a limited space. The pressure in such a globe is produced by the elasticity of the coat and by the volume of its contents. The slightly distensible coat is made up of the cornea and the sclera. The contents consist of a solid lens, uvea and retina, a semisolid vitreous and a liquid blood and aqueous. The distensibility of the cornea and sclera is extremely slight. As given by Duke-Elder from Koster's figures, the increase in the volume of the eye is only 0.007 per cent of the original when the internal pressure is raised from 13 to 70 mm. of mercury.[10]

The influence of the solid and semisolid contents on the intraocular pressure can be seen when the eye is excised or exsanguinated (Duke-Elder). The intraocular pressure then sinks to 10 mm. of mercury and maintains this level until disintegration of the tissues sets in, when it sinks to zero. The residual tension of 10 mm. must therefore be maintained by the slightly elastic sclera and cornea compressing the solid and semisolid contents. The difference in tension between the residual 10 mm. and the normal 18 to 25 mm. of mercury must therefore be maintained by the blood and by the aqueous, which is a derivative of the blood. I may best quote Duke-Elder: [11]

> On allowing the flow of blood to resume in the exsanguinated eye, as, for example, on ligating and then freeing the carotid, or on exsanguination and subsequent perfusion, the vascular channels are refilled, and, since the external coat is only feebly distensible, the entrance of additional fluid involves a rise of pressure. In this way the feeding arteries pile up pressure, and the intraocular pressure therefore rises until a point is reached when an adequate circulation is maintained. At this point the pressure equilibrium is established. . . .

By an adequate circulation is meant a normal inflow and outflow of blood and a normal rate of production and elimination of aqueous.

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8. Duke-Elder, W. S.: Text-Book of Ophthalmology, London, Henry Kimpton, 1932, vol. 1, sect. 4, chaps. 9, 10 and 11.

9. Adler, F. H.: Clinical Physiology of the Eye, New York, The Macmillan Company, 1933, chaps. 13 and 14.

10. Duke-Elder,[8] p. 496.

11. Duke-Elder,[8] p. 495.

The blood and the aqueous are thus the two important structures in the maintenance of the intraocular pressure. The ultimate height of pressure in the eye is determined according to Duke-Elder, and his opinion seems to represent that of most of the investigators on this subject, by two mechanisms, each representing certain physical forces:

1. A hydrostatic-osmotic equilibrium. To continue the quotation from Duke-Elder:[11]

> . . . the ultimate height of the pressure in the chambers of the eye is determined by the hydrostatic pressure in the capillaries minus the difference in osmotic pressure between the aqueous humour and the capillary plasma. If we put the lateral component of the hydrostatic pressure in the capillaries at about 50 mm. Hg, and the difference between the osmotic pressures of the two fluids at 30 mm. Hg, the resultant pressure in the chambers of the eye will be 20 mm. Hg.

2. The safety valve mechanism of the canal of Schlemm. It is pointed out by Duke-Elder that for the maintenance of a continuous circulation there must be a gradient of pressure within the eye. The arterial pressure within the eye must be higher than the capillary, the capillary higher than the venous, and the venous higher than the intraocular pressure. If the intraocular pressure were to rise above the venous pressure, the thin-walled veins would collapse. The average pressure in the intraocular arteries as given by Duke-Elder is 88 mm. of mercury systolic and 64 mm. of mercury diastolic for the retinal arteries and from about 50 to 55 mm. of mercury for the capillaries. The pressure in the vortex veins as given by Lullies[12] is from 9 to 20 mm. of mercury higher than the pressure in the anterior chamber. The pressure in the intrascleral veins, which includes Schlemm's canal, Duke-Elder found to be on the average 1.5 mm. of mercury higher than the intraocular pressure. (A list of pressure readings by various investigators is given by Thiel.[2])

The safety valve mechanism of the canal of Schlemm is conceived as follows: Ordinarily the renewal and the removal of the fluid within the eye are determined by the "dynamic equilibrium existing on either side of the capillary walls between the balancing hydrostatic and osmotic pressures," as mentioned previously. In conditions of raised intraocular pressure, such as normally occurs due to the action of the pulse beat, the respiratory curve and the external muscles of the eye, the pressure in the anterior chamber is temporarily raised above that in Schlemm's canal. Fluid is then forced out of the anterior chamber into Schlemm's canal until the pressure is reduced to normal.

The theory of the safety valve mechanism of the canal of Schlemm is readily acceptable. It is also probable that the aqueous is eliminated through the canal of Schlemm when this is not acting as a safety valve

12. Lullies, H.: Der Druck in den Venen des Scleralkanals, Arch. f. d. ges. Physiol. **199**:471. 1923.

and when the pressure in the anterior chamber is lower than that in the canal.

THE HYDROSTATIC-OSMOTIC EQUILIBRIUM THEORY

I return now to the first mechanism mentioned, the hydrostatic-osmotic equilibrium. This theory, of which Duke-Elder is the chief exponent, is based on a number of experimentally established facts and on a number of assumptions. The established facts are:

1. Proteins of the blood exert an osmotic pressure of about 30 mm. of mercury.

2. The hydrostatic pressure in the intraocular capillaries is from 25 to 30 mm. of mercury higher than the intraocular pressure.

3. It has been found by Donnan that when two solutions, one containing a colloid and crystalloids and the other crystalloids only, are separated by a semipermeable membrane through which the colloid is not diffusible, the crystalloids diffuse through the membrane until an equilibrium is established. The electrically charged colloid retains a number of ions of the opposite charge, so that there is on each side of the membrane an excess of diffusible ions of one charge over those of the opposite charge. When equilibrium is established, however, the product of the concentration of the cations and anions on one side of the membrane is equal to the product of the concentration of the cations and the anions on the other side of the membrane. This is the Donnan theorem.

4. It has been shown by Duke-Elder that the relation of the product of the concentration of cations and anions in the aqueous, which is free from protein, to that in the blood, which contains the nondiffusible protein, corresponds to the Donnan theorem.

5. There is a circulation of fluid within the eye. Fluid moves out of the blood stream into the posterior chamber and through the pupil into the anterior chamber and out again into the blood stream. The latest addition to the proof of such a circulation was made by Friedenwald and Pierce.[13]

6. It has been shown experimentally in animals that when the colloid content of the blood is increased intraocular pressure falls; when the colloid content of the blood is diminished intraocular pressure rises. Similarly, when the blood is made hypertonic with salt solution the intraocular pressure falls; when the blood is made hypotonic the intraocular pressure rises. When the intracapillary pressure in the eye is increased the intraocular pressure rises; when the intracapillary pressure in the eye falls the intraocular pressure also falls.

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13. Friedenwald, J. S., and Pierce, H. F.: The Circulation of the Aqueous, Arch. Ophth. **7**:538 (April) 1932; **8**:9 (July) 1932; **10**:449 (Oct.) 1933; **14**:599 (Oct.) 1935.

The assumptions made on which this theory is based are:

1. "The capillary walls may be considered a semipermeable membrane . . ." (Duke-Elder [14]).

2. Fluid leaving and entering the capillaries moves through the capillary walls and tissues as it moves through an artificial semipermeable membrane.

3. From the experimentally established fact that the proteins of the blood have an osmotic power, it follows that the proteins in the blood exert an osmotic power on fluids on the other side of the capillary walls.

4. The change in the movement of fluid toward the blood stream or away from the blood stream when the colloid content of the blood or the salt content of the blood is either increased or diminished is simply the effect of either increased or diminished osmotic power of the constituents of the blood.

5. The Donnan membrane theorem necessarily plays a role in the body when two solutions with different ionic concentrations, and one of them containing protein, are separated from one another by a membrane.

The Capillary Wall as a Semipermeable Membrane.—Of great importance is the first assumption, that is, that the capillary wall may be considered a semipermeable membrane. It is assumed that the capillary wall behaves in the manner of an artificial semipermeable membrane which is used in the laboratory for the study of the diffusion of solutions. But there is actually no proof that this is so. There is no proof that the capillary wall when taken by itself away from all regulatory influences conforms to the criteria of an artificial semipermeable membrane as used in the laboratory. In fact, it is doubtful whether there is anywhere in the living body any separating membrane which corresponds to a strictly semipermeable membrane. There is a good deal of evidence that the contrary is the case; that the separating membranes in the body are not semipermeable membranes in the foregoing sense; that the movement of fluid through membranes and tissues in the body does not follow the laws of osmosis; that while there is some sort of balance between the fluids on the two sides of a membrane such as the capillary wall, such a balance need not and often does not correspond to the Donnan theorem.

The Movement of Fluid Through Living Membranes.—The fundamental work on the movement of solutions through living membranes has been done by Wertheimer [15] in Abderhalden's laboratory in his

14. Duke-Elder,[8] p. 443.

15. Wertheimer, E.: Ueber irreziproke Permeabilität, Arch. f. d. ges. Physiol. **199**:383, 1923; Ueber die Quellung geschichteter Membranen und ihre Beziehung zur Wasserwanderung, ibid. **208**:669, 1925; Weitere Untersuchungen an lebenden Membranen, ibid. **210**:527, 1925.

studies on the irreciprocal permeability of membranes. This work, which is easily accessible, has evidently not had the consideration it deserves. Similar work has been done on the eye by F. P. Fischer.[16] Wertheimer found that in the living skin of the frog water diffuses more quickly from within outward than in the opposite direction. Under the conditions of the experiment, sodium chloride passed from without inward but not in the opposite direction. Such an irreciprocal permeability he found also with peptone, polypeptide, amino acids and dextrose. The dead skin loses this irreciprocal permeability, and all substances diffuse equally.

Wertheimer also found that the ability of the living skin of the frog to bind water is different on the two sides. The external layer swells markedly in hundredth-normal sodium hydroxide solution, and the inner layer, only slightly; hundredth-normal sulfuric acid solution has the opposite effect. The cations potassium, rubidium, cesium, ammonium, lithium and sodium have different effects on the two surfaces. He noticed similar effects with nonelectrolytes.

Wertheimer found that the movement of water through the living membrane is dependent on the degree of swelling of the two sides when in contact with water. Water enters the living membrane only when the surface in contact with it can take it up. The greater its ability to take up water, the more of it enters the living membrane. Water does not pass through a living membrane as it does through a dead membrane or through an ultrafilter. Instead, the surface layer in contact with the water swells and then gives up its water to the contiguous layer until the water has passed through.

Similar observations were made by Wertheimer on the intestines of the pig.

Wertheimer also found that the two surface layers of living membranes have different ionic concentrations. In the skin of the frog the hydrogen ion concentration is greater in the outer surface layer, while the chloride ion concentration is greater in the inner surface layer. In the lung he found the hydrogen ion concentration greater in the layer near the inner surface, that is, the surface lined with respiratory epithelium, and the chloride ion concentration greater in the layer near the outer, or pleural, surface.

The results of the investigations of Wertheimer show that the rate of movement of water through a living membrane of many layers, such as the skin of the frog or the intestines of the rabbit and the pig, is influenced by the constellation of the electrolytes in the different surface

16. Fischer, F. P.: Untersuchungen über die Quellungsvorgänge und über Permeabilitätsverhältnisse der Hornhaut, Arch. f. Augenh. **98**:46, 1928; Ueber die Permeabilität der Hornhaut und über die Vitalfärbungen des vorderen Bulbusabschnittes mit Bemerkungen über die Vitalfärbungen des Plexus chorioideus, ibid. **100-101**:480, 1929.

layers of the membrane. There is nothing illogical in the assumption that the behavior of other living membranes in the body, such as the walls of the capillaries or the serous membranes, is similarly influenced by variations in ionic composition of their surface layers. The investigations show that the term semipermeable membrane as applied to an artificial or dead membrane cannot be applied to a living one, nor is there here simply a question of the size of the pores, as in a dead membrane. What Wertheimer found for the skin, the intestines and the lungs can without hesitation be applied to the capillary walls, which are multilayered membranes no matter how thin they are. For each capillary wall consists at least of a layer of endothelial cells, a basement membrane and Rouget cells, and each layer may be considered multilayered in a physical sense; in addition, the capillary wall is closely applied to the surrounding tissues, which again are multilayered. It is obvious, then, that a hydrostatic-osmotic balance cannot be the sole factor or even any factor in the movement of fluid through such membranes in the eye or anywhere in the body.

The Movement of Fluid Through the Cornea.—Studies similar to those of Wertheimer have been made by Fischer [16] on the cornea. He studied the diffusion of fluids through the cornea and found there an irreciprocal permeability. Solutions which are able to pass through from without into the anterior chamber do not pass in the opposite direction. Again, substances which can pass from the anterior chamber outward through the cornea do not pass in the opposite direction. Fischer found that it is especially the endothelium of the cornea lining the anterior chamber and the epithelium covering the cornea which determine the passage of solutions. And it is the ability of the lining endothelium and epithelium to take up a solution and pass it on to the stroma which determines whether the solution can pass through the cornea. This characteristic of the endothelium and epithelium is evidently a stable and permanent one and can be changed artificially only by a change in the electrolytic content of the aqueous and tears. It is especially the endothelium in which this characteristic is easily recognizable. For it is this single layer of cells derived from mesoderm, as is the endothelial layer of the capillary wall, which prevents the aqueous from penetrating into the cornea. Damage to the endothelium is immediately followed by the entrance of aqueous into the stroma, an observation made long ago by Leber.

The Donnan Theorem in Relation to the Mineral Content of the Eye.—To determine whether the various structures in the eye are separated by semipermeable membranes and whether the Donnan theorem holds true for them, Fischer [17] made an elaborate study of the

17. Fischer, F. P.: Der Mineralbestand des Auges, Arch. f. Augenh. **107**:295, 1933.

mineral contents of the cornea, lens and vitreous and compared them with the mineral contents of the aqueous and the blood serum. He made similar studies on the vascular tissues of the eye: the sclera, retina, iris, ciliary body and choroid. He used the eyes of pigs and cattle and studied the following cations and anions: sodium, potassium, calcium, magnesium, chlorine, phosphate and sulfate. Fischer [18] came to the conclusion that while the distribution of the various ions between the aqueous and the blood serum may correspond closely to the requirements of the Donnan theorem, no such relation exists between the distribution of ions in the various refracting media. Here there is no Donnan distribution of ions whether in relation to the blood or to the aqueous. For each ion there is a different distribution factor. In his discussion Fischer quoted from a lecture by Donnan delivered in Mainz in 1932, in which he pointed out that in the living tissues, so far as they are "living," thermodynamic equilibria cannot exist because free energy is continuously brought to the tissues in the form of substances which assimilate oxygen. These substances are continually changed into lower as well as higher potentials, while with a thermodynamic equilibrium the free energy reaches a molecular-static minimum. In other words, a system existing in a thermodynamic equilibrium is surely completely "dead." [19] "Disequilibria, not equilibria arise and are maintained as long as life remains."

Fischer stressed the constancy of the mineral composition of the tissues of the eye, which must unequivocally be described as different from their surroundings. In his summary he gave the following conclusion:[20]

> It is concluded from the differences in the mineral content of the tissues and their surroundings, the tissues as well as the fluids of the eye, that conditions of thermodynamic equilibria, ionic distribution according to the Donnan theorem, do not exist and cannot exist as long as the tissues have not died.

In a recent publication Robertson [21] reviewed critically the results of Duke-Elder in regard to the chemical and the physical equilibrium between the blood and the aqueous. His own investigations led him to conclude:

> . . . there is evidence to show that there is no chemical equilibrium between the blood and the aqueous humour. Easily diffusible constituents such as urea, sugar, uric acid, are not present in equal concentrations in blood compared with the corresponding aqueous. . . . There is evidence that a physical equilibrium does not exist between blood and aqueous and that the equilibrium level of the intraocular pressure is not maintained by the hydrostatic force in the capillaries minus the difference in osmotic pressure between the aqueous and the blood.

18. Fischer,[17] p. 305.
19. Fischer,[17] p. 306.
20. Fischer,[17] p. 318.
21. Robertson, J. D.: An Investigation into the Theories and the Formation and Exit of Intraocular Fluids, Brit. J. Ophth. **21**:401, 1937.

With the work of Wertheimer and of Fischer in mind, it is not possible to accept the hydrostatic-osmotic theory for the maintenance of intraocular pressure. Unquestionably intravascular pressure in the eye is of importance, but the balance between hydrostatic and osmotic pressure does not have anything to do with the maintenance of normal intraocular pressure. How then is one to explain the experimental results previously discussed, namely, the movement of fluid into the blood stream and the fall of intraocular pressure following the intravenous injection of colloid substances and hypertonic salt solution? And how is one to explain the opposite effect when the blood is made hypotonic?

MAINTENANCE OF PHYSIOLOGIC VALUES AT A CONSTANT LEVEL

This brings one to the crux of the whole problem, which is that attempts to explain physiologic functions cannot be based on a few purely physical forces without taking into consideration the strictly physiologic element, which is regulatory. Fischer in his summary recognized this difficulty and he stated in the last sentence: "A consideration of the facts found must lead to the conclusion that the mineral contents of the tissues must be held constant within physiological limits by a regulatory mechanism of a special kind." This is physiology, the physiology of constant values. The blood, which is part of the internal milieu in the sense of Claude Bernard, is the phylogenetically evolved optimum for the organism, and its composition is maintained at a constant level within physiologic limits at a considerable effort on the part of the organism. When nonphysiologic solutions of sodium chloride, whether hypertonic or hypotonic, or when nonphysiologic colloids are injected into the blood stream or when the amount of protein in the blood is artificially changed, there is an attempt on the part of the organism to reject the nonphysiologic part and reconstruct the normal composition of the blood. Numerous complex mechanisms are set in motion which eventually cause a return to normal. As a result of these complex processes, fluid moves toward the blood stream to dilute the hypertonic salt solution or the colloid or away from the blood to reestablish the normal concentration when the blood is hypotonic. The movement is in response to reflex regulatory demands, and the movement through the capillary walls does not imply the action of osmotic forces but much more complex ones in the sense in which Wertheimer found them in his studies on multilayered membranes, and these forces are regulated by the phylogenetically evolved adaptive constellation of electrolytes and a central nervous control through neural and hormonal influences. It is, of course, natural that with large amounts of fluid moving outward from the blood stream into the tissues in response to

regulatory activities the eye will get a little of it, or that with fluid moving toward the blood stream the eye will lose a little of its water content.

The experiments which cause a change in the intraocular pressure by changing the composition of the blood can therefore not be used as evidence for the theory of hydrostatic-osmotic balance. It is necessary to come to the conclusion that no theory which has as its foundation the balancing of several physical forces alone can explain the maintenance of normal intraocular pressure.

This brings me to the point just barely touched on by Fischer, that of a regulatory mechanism, and to the wider view of physiologic regulation as pointed out by Claude Bernard in his discussion of the internal milieu, by Haldane [22] in his "New Physiology" and more recently by Barcroft [23] in his "Features in the Architecture of Physiological Function."

The Mechanism for the Maintenance of a Constant Physiologic Value.—The living organism continuously attempts to maintain normal values within narrow limits of variation for those substances which it needs in its economy and for those processes and activities within itself to which it has become adapted. Claude Bernard mentioned water, oxygen, temperature and chemical substances. It is obvious that all processes in the body must be included—the maintenance of normal temperature, blood pressure, blood volume, individual constituents of the blood, general metabolism, metabolism of the individual foodstuffs, digestion and respiration; in short, everything that happens in the body, including, of course, the maintenance of the normal intraocular pressure. While the means of maintenance of the normal value of some of these processes is relatively well understood, little is known of others. It is known, however, that the means which the organism uses to maintain normal physiologic values are in general similar in all processes. These consist of, first, effector organs; second, a central regulating mechanism in the central nervous system, and, third, channels through which the central apparatus influences the effector organs.

1. Effector Organs: The effector organs perform the function for which they are provided. In them function is regulated by means of electrolytes, mainly, the cations hydrogen, sodium, potassium, calcium and magnesium and the anions hydroxide, chlorine and phosphate and probably others. A change in constellation of these electrolytes changes the rate of functioning of the effector organs. An easy example is the

22. Haldane, J. S.: The New Physiology, in Harvey Lectures, 1916-1917, Philadelphia, J. B. Lippincott Company, 1918, p. 21.

23. Barcroft, J.: Features in the Architecture of Physiological Function, London, Cambridge University Press, 1934.

heart beat, which is changed by altering the sodium, potassium and calcium contents of the nutritive fluid.

2. Central Regulation: The regulating mechanism is found in the central nervous system, in which the phylogenetically old vegetative functions have their seat of control in the gray matter surrounding the central canal in the spinal cord and around the third and fourth ventricles in the brain stem. The highest developed centers for these functions are found in the gray matter surrounding the third ventricle, in the hypothalamus. The increasing amount of investigation in this region of the brain has shown that it contains the centers of control of temperature, sleep, water movement, metabolism, blood pressure and even the emotions. Lower down, in the region of the aqueduct, the fourth ventricle and the spinal cord are the controls for the more primitive vegetative functions. While knowledge concerning the centers of regulation for vegetative functions is thus increasing, there are innumerable bodily functions the centers of regulation of which have not been located. That in the economy of the organism they are centrally regulated must necessarily be assumed.

3. Channels of Influence: There are two channels through which the hypothalamic centers can influence bodily function. The first is by means of neural connections with the nuclei of the sympathetic and parasympathetic nervous systems. There are known connections between the hypothalamus and the somatic and visceral centers of the brain stem and spinal cord and through the spinal cord with the sympathetic system.[24] The sympathetic and parasympathetic fibers end in the cells and tissues of the effector organs.

The second channel of influence is through the hormones. It has been shown that nerve fibers running from the hypothalamus to the pars nervosa of the hypophysis influence the secretion of this gland, which, entering the circulation, reaches many effector organs.

MAINTENANCE OF THE NORMAL INTRAOCULAR PRESSURE

The normal intraocular pressure is an evolved optimum for the eye and is maintained within narrow limits of variation. In the light of the previous discussion, I conceive the normal intraocular pressure to be maintained by a unit mechanism consisting of an effector organ, a center of control and neural and hormonal channels by which the center exerts its influence on the effector organ. The whole mechanism acts as a unit to maintain the intraocular pressure at a certain level. With any slight change in pressure, afferent impulses are carried to the center, which reacts by influencing the effector organ to reestablish the normal pressure.

24. Ranson, S. W.: Some Functions of the Hypothalamus, in Harvey Lectures, 1936-1937, Baltimore, Williams & Wilkins Company, 1937, p. 92.

The Effector Organ.—In discussing the individual structures of the eye in relation to intraocular pressure, it was concluded that except for the passive action of the slightly distensible sclera, it is the blood and the circulating fluid in the eye, the aqueous, which maintain the pressure within the eye. There are then two elements which constitute the means of maintaining the normal pressure within the eye: (1) the quantity of circulating fluid, which must be of a constant value within narrow limits of variation, and (2) the quantity of the circulating blood within the eye, which must be of a constant value within similar limits.

1. The aqueous is a watery solution of certain electrolytes and nonelectrolytes which enters the chambers of the eye by passing through the capillary walls and, after circulating in the eye, passes into the venous system, especially by way of Schlemm's canal. The capillary walls in the eye and the wall of Schlemm's canal, no matter how thin, must be considered, like all separating membranes in the body, as multilayered membranes. The passage of fluid through them into the chambers of the eye and out I conceive to occur in a manner similar to that which Wertheimer found in his studies on the passage of solutions through multilayered living membranes. According to this, the entrance and exit of fluid are regulated to maintain the pressure of the eye, first, by the constellation of the electrolytes in the layers of the walls of the capillaries and adjacent tissues; second, by the ability of each layer to take up fluid, and, third, by the extent and size of the capillary bed.

2. The second factor, the quantity of the circulating blood, determines the size of the capillary bed. The vessels which come under consideration are those of the uveal tract, for the retinal circulation is quasi-independent. The quantity of blood in the uvea is dependent on the degree of contraction of the capillaries and on the number of capillaries open at one time. Regulation of the size of the bed is necessary, for it is clear that when it is increased the extent of the capillary wall through which fluid passes is increased, and when it is diminished, the size of the capillary wall is diminished. The means of regulation is conceived to be within the capillary wall itself, with the additional regulation of nerves and hormones. Evidence for this can be found in the work of investigators in this field. In addition to these means of regulation in the wall itself, the blood volume in the capillaries is also kept at normal by the contraction of the arteries and, more important, by the regulation of the outflow from the veins. Diminution in the outflow of blood from the veins is quickly followed by dilatation of the capillaries and, if pathologic, by stasis.

The blood of the uveal tract is finally collected in the vortex veins. Before entering the sclera, each vortex vein dilates to form an ampulla

and then narrows as it enters the emissary canal. In its course through the first part of the emissary canal the wall of the vortex vein is very thin (from 0.004 to 0.006 mm.), but shortly before its exit from the canal it suddenly becomes thicker (from 0.02 to 0.06 mm.). This is due to an increase of connective tissue of the adventitia and also to the addition of circular and longitudinal muscle fibers.[25] The presence of muscle fibers means that the vessel can be narrowed by their contraction, and it is possible that there is, here, an additional mechanism for the regulation of the outflow of blood, a mechanism the importance of which has, perhaps, not been sufficiently appreciated.

In the normal eye the amount of blood remains within definite limits, and only a definite amount enters the arterial system, just enough to keep the capillary bed within the necessary size and extent. It is balanced by the amount of blood leaving the veins, the whole forming a unit considerably independent of the general circulation.

The effector organ for the maintenance of the normal intraocular pressure thus consists of, in addition to the sclerocorneal coat, at least three units: (1) the capillary walls and the walls of Schlemm's canal, the passage of fluid through which is regulated by the electrolytic content of its layers; (2) the amount of circulating aqueous, and (3) the amount of circulating blood. In order to work smoothly and continuously under all conditions such a complex organ must have central regulation. It is hardly conceivable that it can be otherwise. What evidence is there that nerves and hormones control it?

Neural Control.—It has been known for a long time that the vessels in the eye are controlled by fibers from the sympathetic nervous system. The evidence for it is reviewed by Thiel and by Duke-Elder. Histologically, a great network of fibers is found everywhere in the uveal tract, especially around the capillaries. Experimentally, it is hardly possible to separate any effect of the sympathetic fibers on the capillaries from the effect on the large vessels. Stimulation of the cervical sympathetic fibers produces an effect on many structures—small and large arteries, capillaries and the smooth muscle fibers of the orbit. Therefore, the effect of the activity of the sympathetic nerve fiber, in controlling the effector organ for the maintenance of intraocular pressure cannot be gaged from such experiments. But the very presence of fibers points to control by the sympathetic nervous system.

Parasympathetic fibers have not been traced to the capillary walls or to the walls of the larger blood vessels. Experimentally, axon reflexes which cause vasodilatation through antidromic activity followed by an increase in intraocular pressure have been shown to occur. These axon reflexes are mediated through the sensory fibers of the trigeminal nerve.

25. Lauber, H., in von Möllendorff, W.: Handbuch der mikroskopischen Anatomie, Berlin, Julius Springer, 1936, vol. 3, pt. 2, p. 83.

The only real evidence for activity of the parasympathetic fibers is in the work of Velhagen,[26] who found that in the uvea and retina choline and acetylcholine occur, the latter being probably identical with the "parasympathetic stuff" of Loewi.

Hormonal Control.—There is little actual proof that the intraocular pressure is under hormonal influence. The contradictory evidence is reviewed by Thiel,[2] who, however, came to the conclusion that "undoubtedly there is an influence of the endocrine system on the intraocular pressure." In spite of the insufficient evidence, I am convinced that in analogy with other vegetative functions hormonal control constitutes one of the means of control of the normal intraocular pressure.

Central Regulation.—Nothing is known of any center in the brain which controls the maintenance of intraocular pressure. In spite of this, it is necessary to hold to the conviction that there is such a controlling center and to expect that it will eventually be found by future investigators. Such a center would probably be located in the brain stem, in the gray matter surrounding the canal system, where it would be in connection with the sympathetic system, possibly in the hypothalamus, where so many of the centers for vegetative functions are located. The considerations which lead me to assume the existence of such a center, I have already discussed. Mainly, they are: (1) the analogy with other vegetative functions which are known to be controlled by a center in the brain; (2) the inadequacy of the assumption that the action of purely physical forces alone and without regulation can explain the maintenance of such a physiologic constant as the normal intraocular pressure, and (3) the independence of the maintenance of the normal intraocular pressure from other bodily functions. This may also be considered, that the intraocular pressure reacts to some degree with the activities of the body known to be influenced by hypothalamic centers. Especially is this the case with the emotions, which are known to have an effect on the intraocular pressure. It is now known that the center for the integration of visceral and somatic components of emotional expression is in the hypothalamus.[24]

Normal Variations in Intraocular Pressure.—The conception that the intraocular pressure is maintained by a unit mechanism consisting of an effector organ, a center of control and channels through which control is exercised permits at once an understanding of the normal variations in intraocular pressure. The diurnal variations are in close analogy with the diurnal variations in other vegetative functions which are maintained by such mechanisms. It is known that such variations

26. Velhagen, K., Jr.: Zur Frage der vagotropen Substanzen im Auge, Arch. f. Augenh. **105**:573, 1932.

occur in blood pressure, temperature, sugar content of the blood, metabolism and other bodily functions. This conception also permits an understanding of the mechanism of compensation when there is any untoward disturbance in the effector organ. The external ocular muscles exert pressure on the eye and raise the intraocular pressure. An undue rise in pressure causes afferent impulses to be carried to the center, which responds by influencing the effector organ through the nerves and hormones to reestablish the normal pressure. The time which it takes for this compensation to be effected can be studied by exerting a measured amount of pressure on the eye and studying the time it takes for the pressure to return to normal. Such studies have been made (Duke-Elder [27]).

CHRONIC SIMPLE, OR PRIMARY, GLAUCOMA

With the conception of the maintenance of the normal intraocular pressure as outlined, I shall now attempt to explain the pathogenesis of chronic simple glaucoma.

Chronic simple, or primary, glaucoma is a condition in which the intraocular pressure is persistently higher than the optimum for the normal eye and is above that which the eye can withstand for a long time without damage. The condition comes on gradually and insidiously at or after the age of 40 and usually involves both eyes; when it is not checked it eventually leads to blindness. It differs from secondary glaucoma in that it is not secondary to any other illness in the eye and in that there is no apparent cause for its appearance. In this as well as in the age at which it makes its appearance it resembles other diseases which are due to a change in regulation, especially essential arterial hypertension and diabetes.

SYMPTOMS

In the consideration of the symptoms of chronic simple glaucoma it is necessary to separate sharply the ocular changes which are the result of the persistently high tension from the symptoms which appear early in the course of the disease. The main change which is due to the persistently high pressure within the eye is in the posterior segment, the excavation of the disk, with the gradual destruction of one bundle of nerve fibers after another. In the anterior segment the changes are the pushing forward of the lens-iris diaphragm, with the consequent diminution in the depth of the anterior chamber; in the iridocorneal angle the changes are atrophy of the trabeculae, the increased amount of pigment deposit and the narrowing and complete obliteration of the angle and the atrophy of the iris and the synechia between the root of the iris and the cornea. With the destruction of the

27. Duke-Elder,[8] p. 501.

nerve fiber bundles, there is the corresponding loss of the visual field. All these changes, which are the result of a long continued increase in the intraocular pressure, do not give any clue to the pathogenesis of the disease.

Early Changes.—The picture is entirely different in the early stages of the disease. Here there are practically no visible alterations in the eye either in the anterior segment or in the optic disk, and the eye does not differ in appearance from a normal eye. There are no changes in the acuity of vision or in the visual field. There are no subjective complaints in the early stages, with the exception perhaps of headache occurring occasionally in the morning or after a prolonged stay in a dark room. The increase in intraocular pressure is usually found on routine examination when deliberate test is made for it. Subjective symptoms begin to appear with the secondary changes previously mentioned. When the intraocular tension is tested by means of a tonometer, the characteristic changes of the early stages are found, and these are:

1. An increased tension which varies considerably but is most frequently between 30 and 50 mm. of mercury, occasionally higher and sometimes lower. In some cases the tension is within the normal limits most of the day, rising only in the early morning hours.

2. Diurnal variations in tension. In the normal eye these variations are small, from 2 to 3 mm.; in the glaucomatous eye they vary as much as 20 mm., and the daily curve frequently has two peaks, one in the middle of the night and one in the morning.

3. The behavior of the intraocular tension in response to certain tests. These tests are:

(*a*) The administration of caffeine. The tension in the normal eye is not increased, while in the glaucomatous eye there is a decided increase of 10 mm. or more.

(*b*) Compression of the veins of the neck by means of a tourniquet or by lowering the head. This gives a similar increase in the glaucomatous eye, but not in the normal eye.

(*c*) The dark room test. The patient is put in a dark room for an hour. The tension in the glaucomatous eye rises with a variation of from 10 to 40 mm. and returns to the previous reading when the patient gazes at a brightly illuminated wall. The normal eye shows no variations.

(*d*) Massage of the eye. The tonometer with the 10 or 15 Gm. weight is placed on the eye and kept there for a number of minutes. In the normal eye this is followed by a marked drop in tension; in the glaucomatous eye the tension does not go down at all or drops much less than in the normal eye, then quickly returns to its previous reading and frequently exceeds it.

(*e*) Puncture of the anterior chamber. Kronfeld [28] found that when the anterior chamber in the normal eye is punctured with a needle and the aqueous withdrawn the intraocular tension returns to normal in about two hours. In cases of simple glaucoma with normal tension the tension rises to 50 and 60 mm. of mercury after one or one and a half hours.

Age.—In addition to the changed behavior of the intraocular pressure, the age at which chronic simple glaucoma makes its appearance is of importance. Since there are no subjective symptoms in the early stages of glaucoma, statistical reports are not always reliable in giving the actual ages at which chronic simple glaucoma begins to appear. In a recent statistical study, Lehrfeld and Reber,[29] from the Wills Hospital in Philadelphia, found 20 patients with juvenile glaucoma (that is, they were below the age of 35 years) and 1,023 patients with simple glaucoma (40 years of age and over) in a group of 1,876 patients with all varieties of glaucoma.

Characteristics of the Early Stage.—When these changes in the intraocular pressure are reviewed, it is clear that the eye with chronic simple glaucoma in the early stages differs from the normal eye in three characteristics: first, the greater lability or instability of the intraocular pressure during the twenty-four hours of the day, even when the low readings are still within the normal limits; second, the gradual increase in the intraocular pressure above that for the normal eye, and, third, the appearance of these changes, except in the rare cases of juvenile glaucoma, at middle age, that is, at about the age of 40 years or later. The course is somewhat as follows: At about the age of 40 years or shortly before that, the intraocular pressure becomes more labile during the twenty-four hours and shows greater variations. Tests made at this time show greater variations in response than in a normal eye. This lability or instability persists, and within an indefinite period of time, perhaps a few years, the tension not only shows the greater variations but continues to be higher than that of a normal eye. Again in an indefinite period of time the pressure becomes still higher, and with this persisting, secondary changes begin to appear. During the period when the tension is higher there is an additional increment of fluid within the globe, but the amount of fluid entering the eye and leaving it is perfectly balanced. For were it not balanced, a quick and sudden increase of pressure with all its attendant acute changes in the eye would be the result, and maintenance of pressure even at the higher level of from 30 to 50 mm. continuously would be impossible.

28. Kronfeld, P. C.: Das Verhalten des intraokularen Druckes nach Vorderkammerpunktion bei Glaukom, Ztschr. f. Augenh. **7**:48, 1930; Modern Viewpoints as to the Mechanism of Glaucoma, Am. J. Ophth. **12**:480, 1929.

29. Lehrfeld, L., and Reber, J.: Glaucoma at the Wills Hospital, 1926-1935, Arch. Ophth. **18**:712 (Nov.) 1937.

COMPARISON OF CHRONIC SIMPLE GLAUCOMA WITH OTHER DISEASES

The changes in the intraocular pressure as they occur in the early stages of chronic simple glaucoma can be compared with the early changes in other diseases which are the result of a change in regulation.

Observation of the course of essential arterial hypertension will show that around middle age there is a greater lability of the normal blood pressure, with an increase above normal during certain periods and the return to normal during others. Within an indefinite period of time the blood pressure gradually rises and persists at a higher level while keeping its greater lability. Within a further period of time the blood pressure rises still higher, and with its persistence at the high level secondary changes in the arterial system occur. Again, as in cases of chronic simple glaucoma, juvenile arterial hypertension occasionally occurs.

A comparison of diabetes with chronic simple glaucoma will disclose a similar course. Around middle age there is a variation in the sugar content of the blood; it is higher in some periods and returns to normal at others. Within an indefinite period of time the blood sugar is maintained at a higher level, still keeping its abnormal variations. Within a further period of time it is found to be at a still higher level, and when this condition is unchecked and untreated, secondary nutritional changes soon result.

The comparison of chronic simple glaucoma in its early course with the early course of such diseases as essential arterial hypertension and diabetes makes one aware of the fact that chronic simple glaucoma belongs to a group of diseases which are characterized primarily by a change in regulation. There are unquestionably other diseases belonging to this group. In all these diseases there occurs in advancing life a change in the maintenance of certain physiologic values. In essential hypertension it is the maintenance of the normal blood pressure; in diabetes it is the maintenance of the normal sugar content in the blood, and in chronic simple glaucoma it is the maintenance of the normal intraocular pressure. The regulatory mechanism for the maintenance of each of these values first becomes unstable and variable and is gradually so geared or so constituted as to maintain the value at a higher level. The maintenance of such a physiologic value at a higher level, not being the evolved optimum for the organism, eventually causes damage to the organism.

PATHOGENESIS OF CHRONIC SIMPLE GLAUCOMA

In the discussion on the maintenance of the normal intraocular pressure, I came to the conclusion that it is maintained by a unit mechanism which consists of an effector organ in the eye, channels for

neural and hormonal control and a still unknown center in the brain stem. Applying this conception of the maintenance of the normal intraocular pressure to the changes in intraocular pressure in chronic simple glaucoma, I come to the following conclusion: The effector organ in the eye has evidently not lost its ability of allowing the proper amount of fluid to pass into the eye and the corresponding amount of fluid out of the eye. The only change is that there is an increment of fluid in the eye, which is at first temporary and later permanent. The central control and the channels through which control is exercised still function, as seen by the maintenance of a pressure level, although the level is now higher. Instead of maintaining the evolved optimum of intraocular pressure, the unit mechanism has gradually changed its gearing and is now working with the same effector organ and the same channels of control to maintain the intraocular pressure at a higher level. It is not in a particular structure of the eye that one need look for the cause of this alteration in function and not in the effector organ or in the channels of control. The change is either in the center of control itself or in the unit mechanism as a whole. The phylogenetically evolved mechanism for the maintenance of the optimum intraocular pressure is in some persons not the stable mechanism that it should be. At an early period it shows its lack of stability by greater daily variations and by the abnormal responses to tests and finally it loses more and more its ability to maintain the normal pressure.

ETIOLOGIC FACTORS

When one looks for any cause for the loss of stability of the mechanism for the maintenance of the normal intraocular pressure, none is apparent. A review of statistical reports shows that there is no great difference in the incidence of chronic simple glaucoma in the two sexes; that race does not play a role, and that no general or systemic disease of any kind can be held responsible in its causation. There are only two factors which have any meaning in these reports, the age factor, which I have already discussed, and the factor of heredity.

Heredity as a factor in the production of chronic simple glaucoma has long been known, and the occurrence of glaucoma in several members of a family is not uncommon. The type of heredity is that of a dominant one. Plocher [30] reported the occurrence of glaucoma in 17 persons in a family of 46 members in five generations. Von Graefe pointed out the phenomenon of "anticipation," that is, the appearance of glaucoma at an earlier age in the members of the subsequent generations. Loehlein, Lawford, Nettleship and many others have reported cases of hereditary glaucoma, and the literature has been reviewed by

30. Plocher, R.: Beitrag zum juvenilem familiären Glaukom, Klin. Monatsbl. f. Augenh. **60**:592, 1918.

Franceschetti [31] and by Waardenburg,[32] who was able to follow cases of simple glaucoma in two or three generations.

There remains nothing else at the present state of knowledge but to conclude that the loss of stability in the mechanism for the maintenance of the normal intraocular pressure in some persons is the result of an inherited defect already present in the germ plasm.

CONCLUSION

I conceive the pathogenesis of chronic simple glaucoma to be as follows: In certain persons there is as a result of inherited influences a mechanism for the maintenance of the normal intraocular pressure which is constitutionally inferior. This constitutional inferiority is already inherent in the germ plasm and is the cause of the comparatively shorter period of normal activity of the whole mechanism or of its central regulation. In advancing life in most cases, or in early life in the rare cases of juvenile glaucoma, this mechanism as a whole loses its normal stability and is at first able to maintain the normal intraocular pressure only in an incomplete manner. This is shown by the diurnal variations in the intraocular pressure and by the abnormal variations to tests. Within a further indefinite period of time the ability to maintain the normal intraocular pressure becomes still more limited, and the mechanism is now so geared or so constituted as to maintain the intraocular pressure only at a higher level, usually from about 30 to 50 mm. of mercury. The change is either in the mechanism as a whole or in its central regulation. The change in function cannot be considered as being due to an alteration in some individual structure in the eye, whether the sclera, iridocorneal angle, vitreous, uveal blood vessels or nerve supply. With the persistently higher intraocular pressure, which is not the evolved optimum for the organism, secondary changes appear in the various structures of the eye and are found in the later stages of the disease. Chronic simple glaucoma is a disease in which there has occurred a change in the regulation of the maintenance of a normal physiologic value, namely, the normal intraocular pressure.

The pathogenesis of chronic simple glaucoma is comparable to the pathogenesis of certain other diseases in which there occurs in advancing life a change in the regulation of certain physiologic values. Of such diseases, essential arterial hypertension and diabetes are the best examples.

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31. Franceschetti, A.: Die Vererbung von Augenleiden, in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 1, p. 773.

32. Waardenburg, P. J.: Das menschliche Auge und seine Erbanlagen, Haag, Nijhoff, 1932, vol. 7, p. 327.

News and Notes

EDITED BY W. L. BENEDICT

SOCIETY NEWS

British Medical Association, Section on Ophthalmology.—The Section on Ophthalmology of the British Medical Association, which meets in Plymouth, will hold meetings on July 21 and 22. The program for the two days is as follows:

On Thursday, July 21, at 10 a. m., "Co-Operation Between the Ophthalmologist and the Physician in Certain Cases of Visual Loss" will be discussed. The discussion will be opened by Mr. C. B. F. Tivy, Plymouth, followed by Dr. R. N. Craig, Exeter; Prof. J. A. Nixon, Bristol; Dr. C. M. Hinds Howell, London, and Dr. Edward McCulloch, Plymouth.

On Friday, July 22, at 10 a. m., the following papers will be read, followed by a discussion: "Some Special Problems in the Pathology of Glaucoma," Dr. Karl Wessely, Munich; "The Contact Lens Centre—Its Purpose and Policy," Mr. A. Rugg-Gunn, London; "Corneal Transplantation," Mr. J. W. Tudor Thomas, Cardiff; "Vital Staining of the Fundus, Mr. Arnold Sorsby, London; "The Avian Nictitating Membrane," Mr. A. L. Candler and Mr. Norman L. Capener, Exeter. At 2:30 p. m., Miss Ida Mann, London, will give a demonstration of patients fitted with contact lenses.

The president of the section is Sir Stewart Duke-Elder, and the honorary secretaries are, R. W. Payne, 1, The Crescent, Plymouth, and Wing Commander P. C. Livingston, Central Medical Establishment, Royal Air Force, 3-4 Clements Inn, W. C. 2.

Chengtu Eye, Ear, Nose and Throat Society.—The Chengtu Eye, Ear, Nose and Throat Society, the first society in China representing jointly practitioners in ophthalmology and in otolaryngology, was organized at the Eye, Ear, Nose and Throat Hospital at Chengtu, West China, on Dec. 14, 1937. The following officers were elected: Dr. Eugene Chan, president; Dr. Robert A. Peterson, vice president; Dr. C. C. Teng, secretary; Dr. C. R. Peng, treasurer, and Drs. M. L. Hu, K. C. Lang, T. C. Shih and C. C. Teng, editorial committee. The society will meet at the Chengtu Eye, Ear, Nose and Throat Hospital on the second Tuesday of each month at 6 p. m.

The members of this society are derived from the departments of ophthalmology and otolaryngology of the West China Union University at Chengtu, of the National Medical College at Nanking and of Cheeloo University at Tsinan, Shantung, and from practitioners in Chengtu and other centers of West China who are specializing in these fields. The transfer of faculties and students of medical schools from the war zone in East China to Chengtu has created there a unique center of teaching and clinical activity. The transfer has afforded the opportunity of combining the staffs of the departments

of ophthalmology and otolaryngology from these schools with these departments of the College of Medicine of the West China Union University in the continued development of its undergraduate and graduate teaching and clinical program.

Eye Section, Philadelphia County Medical Society.—The scientific meeting of the Eye Section of the Philadelphia County Medical Society was held on May 5, 1938, at the Philadelphia County Medical Society Building. The program consisted of the following presentations: "Drusen of the Optic Nerve," with the exhibition of a case, by Dr. George W. Mackenzie; "Growth in the Vitreous," with the exhibition of a case, by Dr. Mortimer W. Blair, and "Present Status of and Technic for Keratoplasty," by Dr. Ramon Castroviejo, New York.

GENERAL NEWS

Graduate Course in Ophthalmology, University of Rochester.—On Aug. 8-11, 1938, the department of ophthalmology of the University of Rochester School of Medicine and Dentistry will offer at the ninth annual summer graduate course in ophthalmology a comprehensive symposium on refraction and related subjects. While the main part of the course will be devoted to this single important phase of the practice of ophthalmology, sufficient time will be allowed for thorough discussions with the visiting lecturers, who are eminent in the field of medical refraction.

Among the subjects to be presented are: "The History of Spectacles;" "Optical Glass;" "A Practical Review of Physiologic Optics;" "Theories of Accommodation;" "Test Lenses, Correcting Lenses and Optometers;" "Refraction, a Medical Problem;" "Retinoscopy, Types and Methods;" "Subjective Methods of Refraction;" "Astigmatic Dials and Charts;" "Use of the Keratometer;" "Choice of Cycloplegia;" "Benzedrine in Refraction;" "Accommodative States;" "Presbyopia, Hyperopia and Myopia;" "Determination of the Astigmatic Axis with the Cross Cylinders and Astigmatic Dial;" "Choice of Bifocal Lenses;" "Muscle Imbalance;" "Orthoptics in Phorias and Tropias;" "Correction of Aphakia;" "Aniseikonia;" "Telescopic Spectacles;" "Contact Lenses;" "Pitfalls of Refraction;" "Spectacle Fitting," and "Checking the Prescribed Lenses."

A list of the guest lecturers follows: Dr. Walter B. Lancaster, Dr. Avery de Hart Prangen, Dr. S. Judd Beach, Dr. Wendell L. Hughes, Dr. George P. Guibor, Theodore E. Obrig, John G. Paul, Scott Sterling, Frederick W. Jobe and Irving Lueck.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

General

HEREDITARY CHARACTER OF SOME OCULAR DISEASES. E. BIRO, Arch. d'opht. **53:** 685 (Sept.) 1936.

In discussing senile cataract, Biro points out that few ophthalmologists have the opportunity to observe the hereditary nature of senile cataract through several generations. He presents the genealogic trees of two families. The evidence at hand indicates that the hereditary nature of senile cataract is dominant. That of congenital ectopia is also probably dominant. An observation of its occurrence in two generations is cited. Genealogic trees of two families in which cases of pigmentary degeneration of the retina occurred are presented. Reference is made to the relation of the dominant or recessive character to the severity of the condition. The consensus is that when it is recessive complications are more often present; when it is dominant, the retinal changes alone are present. The influence of consanguinity is also discussed. A genealogic tree of a family in which some of the members had congenital ptosis and in which the heredity was dominant is presented. Biro discusses the question of whether the condition is due to a defective muscle supply or to a defective nerve supply, as in some cases it has been shown to be due to one factor and in some to the other. In the cases in which operation was performed by the author, histologic study showed a marked thinning of the levator muscle.

S. B. MARLOW.

General Diseases

HEMOLYTIC STREPTOCOCCUS SEPTICOPYEMIA OF OBSCURE ORIGIN, WITH FAILURE OF SULFANILAMIDE IN TREATMENT. M. VAISBERG, M. Rec. **146:**516 (Dec. 15) 1937.

A white man aged 41, a chronic alcoholic addict who had received intensive malarial therapy for syphilis, sustained a marked proptosis (out and down) of the right eye and numerous contusions and abrasions about the head and jaws as a result of a severe beating.

The right eye was immobile. The conjunctiva was prolapsed above and below and filled with gas bubbles. Vision was limited to perception of light. Tension was 36 mm. of mercury (Schiötz). Prophylactic gas gangrene and tetanus serum was given, and in eleven days practically all the ocular symptoms cleared. During this time the left side of the lower jaw was slightly painful and swollen, but an injection of milk apparently helped. Dental care was instituted for the badly diseased teeth.

On the sixteenth day signs of an acute severe general infection developed, with complete loss of vision in the left eye within twenty-four hours. The pupil was dilated at maximum, and the cornea was clouded.

There was excruciating pain, with a tension of 35 mm. of mercury (Schiötz). A large bleb was present on the right heel. There was a foul discharge from the fistula in the left side of the lower jaw. Since the complete picture had not yet developed, an operation for glaucoma was performed to relieve the excessive tension of the left eye; the bleb on the right heel was incised, and a tooth was removed at the site of the infection in the jaw. Sulfanilamide was administered in doses of 90 grains (5.85 Gm.) daily and continued until the patient died.

Despite therapy, infected embolic infarcts developed in the toes and fingers and became progressively worse. Culture from the bleb showed a hemolytic streptococcus. Twenty thousand units of antistreptococcus serum was given daily in addition to the sulfanilamide, but without any benefit. Eight days after the onset of the septicopyemia, the patient died. Autopsy showed a normal brain, an embolus of the central artery of the left retina, osteomyelitis of the left side of the lower jaw, pyopericardium, and infected infarcts in the spleen and kidneys. The organism cultured from the pus in the pericardium was a hemolytic streptococcus.

AUTHOR'S ABSTRACT.

OCULAR MANIFESTATIONS OF BACILLARY DYSENTERY. TOULANT and SARROUY, Arch. d'opht. **53**: 523 (July) 1936.

Bacterologic uncertainty makes it difficult to individualize the ocular lesions due to the different varieties of dysentery. The authors discuss the clinical aspect of the ocular lesions occurring in bacillary dysentery and their pathogenesis. Scleroconjunctivitis is the most common lesion. This condition often appears with lesions of the joints and has its onset late, from ten to twenty days after intestinal symptoms have appeared, and in cases of mild involvement even after these symptoms have cleared. It is self limited, treatment as a rule being unnecessary. Iritis and cyclitis are much more rare and occur later than conjunctivitis (in from twenty to forty days). The authors report a case in detail. Other complications which have been reported include corneal ulcer, serpiginous ulcer, motor paralysis, choroidal hemorrhages and dacryoadenitis. Three hypotheses, i. e., the direct action of the dysentery bacillus, the effect of toxin and the effect of secondary infection, have been proposed to account for these complications.

S. B. MARLOW.

Glaucoma

EXFOLIATION OF THE SUPERFICIAL LAYER OF THE LENS CAPSULE (VOGT) AND ITS RELATION TO GLAUCOMA SIMPLEX. E. HÖRVEN, Brit. J. Ophth. **21**: 625 (Dec.) 1937.

Hörven states that the clinical picture of exfoliation consists of flakes on the pupillary border, a central disk and a peripheral band on the capsule of the lens and changes in the zonule of Zinn.

The flakes never occur as an isolated phenomenon but appear together with an alteration in the capsule. The flakes are therefore of a secondary character, having originated from the surface of the capsule and having become detached spontaneously or rubbed off during the movements of the iris and deposited on the pupillary border.

The central disk has the size and shape of the pupil and is of uniform structure. The peripheral band is the most prominent feature. It lies mainly in the outer third of the distance from the anterior pole of the lens to the equator, but it may be situated elsewhere. The band is invariably present in all patients who have exfoliations.

The appearance in all cases of exfoliation is similar. According to the author, the zonular changes are of a constant nature and form an integral part of the clinical picture of exfoliation.

The fibers of the ligament are covered with scurflike flakes. Hörven has examined 150 patients with the slit lamp, all of whom had been operated on for glaucoma by iridencleisis with meridional iridotomy after the method of Holth. One hundred and twenty-eight (85.33 per cent) had typical exfoliation of the capsule of the lens. Of 43 patients with glaucoma, 40 (93 per cent) showed exfoliation before operation.

Hörven is more and more inclined to believe that exfoliation offers an excellent basis for the classification of glaucoma. Glaucoma simplex must be regarded as identical with glaucoma capsulare, while exfoliation does not occur in cases of acute glaucoma or of chronic inflammatory glaucoma. Exfoliation was found in 2 of 67 inmates of old age homes without signs of glaucoma, in 1 of 34 bodies of elderly persons in the dissecting room, in 10 of 55 patients with cataract and in 5 of 30 patients without cataract or glaucoma.

It does not seem possible to find any other explanation than that senile exfoliation of the capsule of the lens must in some way or other be the cause of the form of glaucoma which is called glaucoma simplex.

W. ZENTMAYER.

FUCHS' EPITHELIAL DYSTROPHY OF THE CORNEA AND CAPSULAR GLAUCOMA. J. MALBRÁN, Arch. de oftal. de Buenos Aires **12:** 441 (July) 1937.

In a patient who at no time showed any signs of hypertension of either eye there was observed "exfoliation of the lens capsule" (capsular glaucoma) of the right eye and "epithelial dystrophy of the cornea" (Fuchs) of the left eye.

Malbrán reviews at length the scientific knowledge in relation to these processes up to date and the connection of both with glaucoma conditions.

C. E. FINLAY.

Hygiene, Sociology, Education and History

NATIONAL POLICY ADOPTED IN A TROPICAL COUNTRY FOR THE PREVENTION OF BLINDNESS. A. F. MACCALLAN, Brit. J. Ophth. **22:** 65 (Feb.) 1938.

This article contains much interesting data concerning the causes of blindness in tropical countries, especially Egypt. The statistics concerning the ophthalmic hospitals in Egypt for 1930 show that of 838,625 patients examined the number of those blind in one or both eyes was 60,157. Conjunctivitis, primary glaucoma, cataract and endogenous iritis were the principal causes. It is particularly to be noticed that in

tropical countries blindness is much more common among the rural population than among urban dwellers. The value of the ophthalmic campaign in Egypt may be judged by the percentage of persons blind in one or both eyes who come to the hospitals and crave treatment. In 1911 the percentage of cases of blindness, including those due to cataract, was 19.2, and in 1935 it was 6. The author rightly believes that the system adopted for the ophthalmic campaign has been justified by the results.

W. ZENTMAYER.

Injuries

RARE CASE OF AN INTRAOCULAR FOREIGN BODY. J. GALLINO, Arch. de oftal. de Buenos Aires **12:** 403 (June) 1937.

A case of a foreign body lodged in the optic disk is reported. The foreign body was observed ophthalmoscopically and localized by the Sweet method.

Gallino has found only one other similar case in the literature, that reported by von Szily, which he reproduces.

C. E. FINLAY.

INTRAOCULAR FOREIGN BODY: RETINOGRAPHIC HISTORY. J. L. PAVIA, Arch. de oftal. de Buenos Aires **12:**433 (July) 1937.

A case of an intraocular foreign body lodged 2 prism diopters below and external to the macula is reported. Its presence was tolerated without reaction for eighteen years. The different stages in the development of the ophthalmoscopic lesion were illustrated before the Argentine Society of Ophthalmology by a series of stereoretinograms.

C. E. FINLAY.

Lids

MECHANISM AND PATHOGENIC VALUE OF VON GRAEFE'S SIGN. J. VOISIN, Ann. d'ocul. **174:** 666 (Oct.) 1937.

The study of the movement of the upper lid in downward gaze may bring into evidence two kinds of pathologic changes. One change is rarely encountered. The author proposes to call it a paradoxic raising of the upper lid, and Fuchs, in the course of repair of certain paralysis of the third nerve, has described it under the name of the pseudo sign of von Graefe. The other change is the sign of von Graefe, which is generally looked for in cases of exophthalmic goiter. This sign, contrary to the classic opinion, may be met with in the course of syndromes in which the sympathetic nervous system is not manifestly the cause.

In order to appreciate the value of von Graefe's sign, one should first study the physiologic mechanism of the associated movements of the globe and upper lid on downward gaze, as it is this movement that determines the symptom considered. The mechanism of these movements is far from being completely solved.

The presence of von Graefe's sign permits of a diagnosis of hypertonia of the muscles of the lid, but it does not give any information as to the cause of this hypertonia.

S. H. MCKEE.

Methods of Examination

THE TESTING OF FITNESS FOR NIGHT FLYING: THE LIGHT SENSE. C. E. FERREE and G. RAND, Am. J. Ophth. **20:** 797 (Aug.) 1937.

Ferree and Rand present the following summary:

"In determining fitness for night flying, important functions to be tested are (*a*) the ability to see at night and at low illumination and the effect of dark adaptation on this ability and (*b*) the amount and speed of dark adaptation. Of these latter functions, speed of adaptation seems to be more important than amount. The night flyer needs especially the power to change his vision quickly from the illuminated cockpit and instrument panel to the outside world and back again. Normal or better-than-normal sensitivity in light adaptation is, of course, also important. The eyes that are needed for night flying are the best of what might be called the normal group; that is, of those that have both good dark and good light vision. More important than speed and range of adaptation, however, is the place in the scale of sensitivity in which the change occurs. That is, it is quite possible that a candidate might have a good range and speed of adaptation and still a poor power to see at low illumination both at the beginning and at the end of the period of dark adaptation. Such a person would be obviously unfit for night flying. To be fit for night flying the candidate must have a normal or better-than-normal rating in power to see at low illumination at the beginning of the period of dark adaptation and throughout its entire course from beginning to end.

"Tests are described for the light minimum under light and dark adaptation and for determining the amount and speed of adaptation. A special test of fitness for night flying is also recommended which is sufficiently quick and convenient for use in routine testing and a procedure is discussed for proving the significance of the test, and for determining the critical values to be used in accepting and rejecting candidates for night flying. A suitable instrument is recommended and its advantages for making the test are briefly discussed."

W. S. REESE.

ORBITAL EMPHYSEMA AS A DIAGNOSTIC MEASURE. M. ORIBE, Arch. de oftal. de Buenos Aires **12:** 458 (July) 1937.

Having observed that the eyeball showed more distinctly in roentgenograms made in cases of fracture of the superior maxillary bone with orbital emphysema, the author conceived the possibility of using artificial orbital emphysema for diagnostic purposes in a manner similar to that with which retrorenal emphysema is used in the diagnosis of certain renal lesions.

Oribe employs a 10 cm. needle, with which he penetrates the orbit at the external palpebral angle, taking care to keep clear of the conjunctival cul-de-sac. Before he injects any air, he first draws out the piston to make sure that he has not entered a blood vessel; he then injects the air slowly at distances of 1 cm., gradually withdrawing the needle. The total amount of air injected varies from 15 to 30 cm.

He reports a case of orbital tumor in which roentgenograms were taken in Blondeau's position before and after the injection of air and in an occipital position; the tumor showed excellently after the injection.

He refers to the use of injections of air, iodized poppyseed oil 40 per cent and diodrast (Katz and Ledoux) into tenon's capsule for diagnostic purposes by different authors since 1927.

C. E. FINLAY.

Neurology

THE DIAGNOSIS OF DISTURBANCES OF THE OPTIC CHIASMA AND THE SELLA TURCICA. E. VELTER, Arch. d'opht. **53**: 593 (Aug.) 1936.

The salient points in the diagnosis of lesions in the area of the optic chiasma and the sella turcica are briefly discussed by the author. He emphasizes, however, the necessity of recognizing the possibility that only isolated signs may be present in a given case and that if early diagnosis is to be made one should not wait for complete evidence. The lesions which occur in this area are divided into those of tumor and those due to inflammation or trauma. The intrasellar tumors consist of chromophobe adenomas, acidophilic and basophilic adenomas and craniopharyngiomas. Illustrative cases are cited. The retrosellar, or subsellar, tumors are composed mainly of malignant growths of the sphenoid, in which there is no characteristic hypophysial syndrome. The suprasellar tumors include the large tumors of Rathke's pouch which have extended outside the sella turcica; the meningiomas, median, lateral and anterior, and tumors of the third ventricle. Reference is made to the possible occurrence of aneurysm, cholesteatoma and gliomas. In the second group the author discusses trauma briefly. Cases of syphilitic meningitis and arachnoiditis are reported. The conclusion drawn from a diagnostic point of view is that a disease of this area is not always a tumorous condition; that drawn from a therapeutic standpoint is that radiotherapy is the treatment of choice for adenoma when it is diagnosed early. This treatment is of no value for the other lesions and may be distinctly harmful in that surgical treatment later may be more hazardous. Each form of treatment, medical, radiologic and surgical, has its indication and should not be used indifferently.

S. B. MARLOW.

HOMOLATERAL HORNER SYNDROME IN EXPERIMENTAL LESIONS OF THE OPTIC THALAMUS. A. GARCIN and M. KIPFER, Compt. rend. Soc. de biol. **126**: 864, 1937.

Sympathetic ocular disturbances have been occasionally observed in persons with thalamic lesions. In a case recently reported, Garcin emphasized the homolaterality of the ocular phenomena. With bipolar electrolysis the authors were able to reproduce like syndromes in dogs, by means of lesions limited to the thalamic muclei. Positive results occurred when the lesion was located between the chiasm and the mammillary tubercles, affecting particularly the anterior portion and the external nucleus of the optic thalamus. The ocular disturbances occurred tardily after a latent period of five or more days. The Horner syndrome hence can be produced independent of the hypothalamus and the cerebral peduncle.

J. E. LEBENSOHN.

SPASMUS NUTANS. G. ÖSTERBERG, Acta ophth. **15:** 457, 1937.

Spasmus nutans is an infantile disease, usually beginning at an age of from 6 to 12 months but never after the third year. The classic signs are nystagmus of the head, torticollis and nystagmus of one or of both eyes. These signs disappear during sleep. The ocular nystagmus is intensified if the head is held still. Complete recovery occurs after a couple of years. Nystagmus of the head alone may occur and is suggestive of spasmus nutans, while if ocular nystagmus is the only sign present, as it may be, care must be taken to differentiate the condition from congenital or early acquired amblyopia associated with strabismus or refractive anomalies. Labyrinthine disease must be excluded by functional tests. The author gives short reports of 4 true and of 2 spurious cases of spasmus nutans.

O. P. PERKINS.

Ocular Muscles

SITE AND NATURE OF THE PROCESS OF IMAGE INHIBITION IN CASES OF STRABISMUS. H. HARMS, Arch. f. Ophth. **138:** 149 (Oct.) 1937.

To analyze the process of image inhibition, or suppression, in cases of strabismus, two methods were used, namely, binocular perimetry and determination of the pupillomotor sensitivity of central and peripheral retinal areas. The perimetry was done principally on the Bjerrum screen, with the patient wearing a red glass over one eye and a green glass over the other eye. A large red target, which was visible to one eye as a colorless bright spot and invisible to the other eye, was used to detect scotomas in the former eye. With a white target it was determined to what extent each eye actively contributed to the binocular field. The patient, then, saw a greenish or a reddish spot if the particular area of the field was "covered" chiefly or exclusively by one eye; if both eyes were active in the particular portion of the field, the target appeared as a mixture of red and green or as two targets. In addition to this subjective method, the author used a method of objective perimetry, namely, the determination of the pupillomotor sensitivity of the retina. Through a diaphragm with central and peripheral slots arranged in the shape of an isopter, central and peripheral retinal areas of equal size were alternately illuminated and the pupillary reactions observed. By dimming the light that fell on the central retinal area, the pupillary reactions occurring on illumination of the central and peripheral retinal areas were made equal. The relation between the two intensities of light which produced equal pupillomotor effects, the so-called equi-pupillomotor quotient (central: peripheral intensity of light), could be determined with a fair degree of accuracy. In normal controls the quotient varied greatly but remained always smaller than 1; in patients with central scotomas due to lesions of the infrageniculate portion of the visual pathway, the quotient was greater than 1.

Patients with strabismus were tested with these two methods. Those with unilateral strabismus, amblyopia and inability to fixate usually showed in the squinting eye a central scotoma and always suppressed impressions received by the retinal area of the squinting eye which corresponded to the macula of the other (normal) eye. The latter phenomenon prevailed bilaterally, that is, in each eye of persons with

alternating strabismus and anomalous retinal correspondence. In persons with alternating strabismus and normal retinal correspondence, all impressions received by the squinting eye were inhibited. In persons with central scotoma the equipupillomotor quotient was greater than 1, a fact which indicated that the pupillomotor sensitivity of the retina in the central region was also reduced. This parallelism between sensory and pupillomotor sensitivity suggests that the process of image inhibition in cases of strabismus takes place in the infrageniculate portion of the visual pathway. Since the inhibition is never hemianoptic, the process must be infrachiasmal. In the infrachiasmal portion of the visual pathway only the retina contains ganglion cells and therefore is the logical site of the process of image inhibition. The accuracy with which the inhibition takes place in each eye simultaneously or alternately justifies the assumption of a central control (probably by the suprastriate fusion center) of the peripheral inhibitional process.

Amblyopia ex anopsia is not the result of an anopsia but constitutes, according to Harms, the most severe and permanent form of image inhibition in cases of strabismus. All the sensory peculiarities which have been described in cases of strabismus are phenomena of adjustment to the abnormal position of the eyes and can, therefore, not be the cause of strabismus.

P. C. KRONFELD.

Operations

FROM OPTICAL TRANSPLANTATION TO THE TREATMENT OF KERATITIS AND CERTAIN CUTANEOUS DISEASES BY TISSUE TRANSPLANTATION. V. P. FILATOV, Vestnik oftal. **11**: 295, 1937.

This article lends itself to abstracting with difficulty. The basis for clearing of the corneal transplant and also the action of additional keratoplasty on the opaque transplant are discussed in detail. Cases of tuberculous, herpetic, pannus and interstitial syphilitic keratitis are reported in which additional keratoplasty favorably influenced the inflammatory process of the cornea. Photographs of 6 patients are presented to illustrate the marked improvement in the cornea after the operation. Filatov believes that additional transplantation stimulates and increases the resistance of the corneal cells to the inflammatory agent and that a biochemical process of an autocatalytic nature takes place in these cells. The treatment of keratitis by transplantation is successful when both the tissue of the host and that of the guest are of the same histologic and zoologic type.

Based on this fact, Filatov made an attempt to treat lupus vulgaris of the skin by transplantation of the skin of a cadaver. Since it can be preserved for several days, Filatov believes that it has an advantage over "fresh" skin, as it will not absorb as quickly as the former. Three cases are reported in which the skin of a cadaver was transplanted on patients with lupus vulgaris of the face, nose and hands and in which the transplant "took" and acquired elasticity. Thus Filatov believes that homoplastic transplantation of various conserved tissues of the corresponding histologic type is a powerful therapeutic agent but that in general this treatment requires further study. Photographs accompany the case reports.

O. SITCHEVSKA.

Orbit, Eyeball and Accessory Sinuses

PARASITIC PSEUDOTUMORS OF THE ORBIT. C. PASCHAFF, Arch. d'opht. **53:** 657 (Sept.) 1936.

Parasites invading the orbit produce two kinds of pathologic reactions: inflammatory, leading to suppuration, and neoplastic, such as pseudotumors. In a section on orbital tumors due to zooparasites the author discusses the lesions produced by Protozoa and by the platyhelminths and nemathelminths. A case of syphilitic gumma is cited to illustrate the first type of lesion, and a case of echinococcosis and 2 unusual cases of cysticercosis, to illustrate the second type, reference being made to trichinosis and filariasis as examples of infection due to nemathelminths. In a second section dealing with orbital tumors due to phytoparasites a case of symmetric tuberculoma is presented to illustrate a subgroup of bacterial pseudotumors, and a case of actinomycosis, to illustrate orbital infection due to Discomyces. In the latter case the author found an organism not unlike Streptothrix farcinica bovis, for which he suggests the term Streptothrix actinomyces. The interest in such cases lies in the fact that the condition tends to engulf the optic nerve and produce atrophy. Surgical extirpation has been successful in some instances.

S. B. MARLOW.

OPHTHALMIC SEQUELS OF THE RADICAL CURE OF FRONTOETHMOID SINUSITIS. P. HALBRON, Arch. d'opht. **53:** 861 (Dec.) 1936.

This report is based on a study of 48 patients operated on for frontoethmoid sinusitis or pansinusitis. Classically the motor defect which results is involvement of the superior oblique muscle, but a study of the cases presented here indicate that other muscles may be affected. At least six months should elapse before surgical correction is attempted, as many patients recover without it. The principal lacrimal complication is epiphora, which often clears with the subsidence of the subcutaneous inflammation. Edema of the lids is frequent. Sometimes abscesses occur. Although purulent conjunctivitis is seldom seen, corneal disturbances have been observed. Such disturbances seem to arise from defective bandaging, which leaves the eye uncovered. In most instances closure of the lids results in cure in a few days. As a result of this study, the author points out that when an operation is done the incision must avoid the lacrimal sac, that care must be taken not to injure the pulley, and that after operation a dressing which closes the lids should be applied.

S. B. MARLOW.

Retina and Optic Nerve

RETROBULBAR NEURITIS IN CASES OF SEROUS MENINGITIS. E. PUSCARIU, Brit. J. Ophth. **21:** 599 (Nov.) 1937.

Of 10 cases of retrobulbar neuritis, in 2 that condition was associated with serous meningitis.

The first case was that of a woman aged 23, who lost her sight in four days, after marked orbital and ocular pains. The blindness persisted fifteen days and receded somewhat at the moment of admission

to the clinic, when the patient was able to count fingers at 20 cm. with the right eye and at 40 cm. with the left eye. The visual field of the right eye could not be explored. There were a central scotoma in the left eye and decolorization of the nasal half of the optic disk. The Wassermann reaction of the blood was positive, and there was a slight rachidian hypertension. The spinal fluid was normal, except for a positive Wassermann reaction. After protein therapy and antisyphilitic therapy, sight was gradually restored, reaching 2/3 in the left eye and 1 in the right eye after four months, with disappearance of the central scotoma. The spinal fluid contained only 6 lymphocytes. The punctures were always well supported.

The second case was one of retrobulbar optic neuritis in a youth aged 19. He admitted a certain excessive use of tobacco and alcohol. At the beginning no signs of the disease were present ophthalmoscopically. From Jan. 4, 1933, to Feb. 28, 1934, seven lumbar punctures were done. The lumbar tension (with the patient seated) varied between 50 and 80 cm., except at the last examination, when it was only 25 cm. Chemical and cytologic examinations did not reveal any abnormality. The punctures were in general well supported and gave some relief. On admission to the clinic the patient could count fingers at 60 cm. with the right eye and at 1 meter with the left eye. Vision improved at the last examination to 1/8 in the right eye and 1/6 in the left eye. The visual fields for red and green were restored, and the central scotomas were much reduced.

The practical conclusion to be drawn from the author's observations is that in dealing with retrobulbar neuritis one should think also of serous meningitis and practice lumbar puncture. In addition to its diagnostic value, this procedure affects favorably both the general cerebral symptoms and the ocular manifestations.

W. ZENTMAYER.

GRÖNBLAD-STRANDBERG SYNDROME (ANGIOID STREAKS OF THE RETINA AND PSEUDOXANTHOMA ELASTICUM) AND ITS RELATION TO DISEASES OF MESENCHYMAL TISSUES. A. FRANCESCHETTI and E. L. ROULET, Arch. d'opht. **53**: 401 (June) 1936.

The authors conclude that from the ocular standpoint this syndrome is characterized by lesions which are encountered in mesenchymal tissues in general. Two cases are reported in detail and the literature is reviewed to lend support to this idea. In the first case there were typical ocular and cutaneous changes, which are illustrated by photographs and colored drawings of the fundi. The patient's parents were consanguineous. In the second case the picture of the fundus was characteristic, but there were no cutaneous manifestations. Diabetes was present. The authors suggest that the name syndrome of Grönblad and Strandberg makes it possible to include under this condition those cases in which there is pseudoxanthoma with chorioretinal changes but no angioid streaks as well as those cases in which angioid streaks are present but pseudoxanthoma is lacking. They point out that there is some reason for believing that this condition is hereditary and recessive in type. Reference is made to theories proposed to account for the changes present. Tuberculosis as a provocative factor, Paget's disease,

Osler's disease and the occurrence of hemorrhages are all considered. The authors believe that diabetes is more commonly present than has been reported. A 6 page bibliography is appended.

S. B. MARLOW.

RECURRENT RETINOVITREOUS HEMORRHAGES IN THE YOUNG. P. BONNET, L. PAUFIQUE and L. SARRAZIN, Arch. d'opht. **53:** 849 (Dec.) 1936.

The authors first review historically the reports which have been published on this condition and briefly discuss the etiologic factors suggested. The question as to whether the hemorrhages are due to diapedesis or rupture of a vessel is raised. Bonnet, himself, favors the idea of a slow breaking of the wall of the vessel. The question of tuberculous periphlebitis is discussed in relation to it. A case is described in which changes were observed ophthalmoscopically prior to the appearance of hemorrhage, forecasting the hemorrhage, and at the time of the hemorrhage. The late results of the hemorrhage were also noted. Colored plates illustrate the case report. Active tuberculosis was shown to be present clinically and bacteriologically. After extensive hemorrhage into vitreous had cleared, definite areas of periphlebitis were found and are depicted. While many causes for this condition can properly be considered, most important is the presentation of complete clinical or anatomic observations made in cases of this kind.

S. B. MARLOW.

RETINITIS CIRCINATA IN A HIGHLY DEVELOPED STAGE: REPORT OF A CASE. M. HANDMANN, Klin. Monatsbl. f. Augenh. **98:**618 (May) 1937.

Handmann reports a case of retinitis circinata in a woman aged 49. He observed the patient for five years, from the initial stage to the development of an extensive white degenerated area in the retina. The patient was otherwise perfectly healthy. The condition in the eye represented the final stage of retinitis circinata, if based on Fuchs' first description, because the appearance of the retina deviated in many respects from that in exudative retinitis. The diagnosis could be ascertained because the process was observed clinically from the beginning to the end. This case proves again that retinitis circinata which is not associated with any complications may lead to extensive massed exudations in the posterior layers of the retina in the course of several years.

K. L. STOLL.

ROENTGEN THERAPY OF RETINAL GLIOMA. H. SCHEYHING, Klin. Monatsbl. f. Augenh. **98:** 756 (June) 1937.

The right eye of a boy aged 4 was enucleated for glioma, and the preoperative diagnosis was corroborated by histologic examination in November 1927. Shortly afterward the left eye was treated with roentgen irradiation. Vision was 5/5 in the beginning of 1931, but radial opacities in the lens were observed by the end of that year. The

cataract was extracted by another oculist in 1935, and a discission of a secondary cataract was performed by the author in December 1936. Illustrations show the difference in the appearance of the fundus in 1931 and in February 1937. The tumor, located above the disk, was greatly reduced and showed deposits of lime. Scheyhing was surprised to observe atrophy of the choroid surrounding the tumor and extending into the region of the macula; he considers it as a lesion due possibly to irradiation.

Four questions of importance are answered by the author in connection with this case. The diagnosis of glioma was confirmed by the histologic examination of the right eye and by the ophthalmoscopic examination of the left eye. Recovery actually occurred, as no recurrence was observed within ten years after roentgen irradiation. A case of the same duration of observation was reported by Heine.

It is impossible to decide whether recovery was the result of irradiation or if it occurred spontaneously, as reported by a number of authors in whose cases the eyeball remained unaltered and vision was preserved. Scheyhing thinks that a glioma as small as that observed in the left eye of his patient may have offered a tendency for spontaneous recovery but that this tendency may have been stimulated by irradiation. Detrimental lesions as a result of irradiation were not observed, but slight changes in the conjunctiva and the cataract developed. The child received small doses, of from 30 to 50 per cent, during the first two years of his treatment, and two large doses, of 80 and 100 per cent, respectively, during the third and last year.

K. L. STOLL.

REPORT OF A CASE OF VERY REMARKABLE VASCULAR CHANGES IN THE FUNDUS. T. KIKKAWA, Acta soc. ophth. jap. **39**: 16 (Feb.) 1935.

Takayasu in 1908, Nakajima in 1921 and Uchino in 1930 reported cases of this unusual alteration of the retinal vessels, though the condition has not been formulated as an independent disease picture. These cases showed in addition other anomalies of the circulatory apparatus. In the case reported, that of a girl 19 years of age, the changes were as follows: The condition occurred in both eyes. It was accompanied by marked diminution of vision and terminated in cataract. The numerous looplike communications between the arterial and venous blood vessels of the retina were rounded, fusiform or cylindric dilatations of the vessels, forming a ring around the disk, without inflammatory symptoms. The other circulatory anomalies were absence of the arterial pulse, the impossibility of measuring the blood pressure, and a bruit over the jugular vein.

A. KNAPP.

Trachoma

CULTURE OF RICKETTSIAS OF TRACHOMA IN VITRO. POLEFF, Arch. d'opht. **53**: 882 (Dec.) 1936.

The author describes in detail the methods used in culturing trachomatos tissue. By this method he has been able to isolate and culture rickettsias of trachoma and to carry the organisms through five transplants. Some evidence is put forward to show the close relation

between these rickettsias and the inclusion bodies of Prowazek and Halberstaedter. The author leaves the etiologic and specific nature of these organisms for further consideration until more immunologic experiments have been completed.

L. B. MARLOW.

AUTOHEMOTHERAPY FOR TRACHOMATOUS PANNUS. B. P. LERNER, Vestnik oftal. **10:** 841, 1937.

Thirty-eight patients suffering from trachomatous pannus were treated by autohemotherapy. For fourteen of these, this treatment was combined with local therapy (the use of atropine or silver nitrate, expression, etc.). A subcutaneous injection of 10 cc. of blood was given every other day, until fifteen injections had been given. In cases of recent pannus, infiltrates or ulcers of the cornea, both the combined therapy and autohemotherapy alone gave good results, but there was little response to the treatment in cases of long-standing pannus.

O. SITCHEVSKA.

Tumors

CONCERNING CONDITIONS SIMULATING AN INTRAOCULAR TUMOR. B. A. KLIEN, Am. J. Ophth. **20:** 812 (Aug.) 1937.

Klien reports the clinical findings and pathologic changes in 4 cases of a condition simulating intraocular tumor and gives the following summary and conclusions:

"In three different conditions suspicion of an intraocular new growth was raised by the following clinical symptoms: 1. A more or less extensive retinal detachment. 2. A rise in intraocular tension that could not be controlled by medication and that in two of three eyes had a fluctuating character. 3. Circumscribed, poor transillumination of the eyeball in one or more places in three of the eyes. 4. The discovery of rigid, circumscribed, dark-appearing elevations which could not be interpreted correctly in two eyes in which the fundus was visible.

"In regard to the first symptom, the retinal detachment was twice due to Coats's disease, once it followed a small disinsertion of the retina in an apparently slightly myopic eye, and once it was preceded by a spontaneous choroidal detachment.

"As to the second symptom, the secondary glaucoma that was present in three of the four eyes was due to an obstruction of the chamber angle, consisting of iritic adhesions in two eyes. In the third eye, which had a wide-open angle, it was possibly due to the nature of the exudate that blocked the spaces of Fontana temporarily between the hypotonic periods.

"In connection with the third symptom, the areas of poor transillumination in one of the eyes corresponded to histologically found areas of hemorrhage in retina and vitreous. The dullness in the two other eyes may have been due to partial dispersion of the penetrating rays by the smooth, round posterior surface of the cyst and the equally smooth, pigmented and rigid, curved surfaces of the choroidal detachment.

"Finally, the dark appearance of the circumscribed elevations ophthalmoscopically was an optical phenomenon in the eye with the large cyst and due to the pigment epithelium of retina and ciliary body in the eye with the choroidal detachment.

"I do not wish to give the impression that the enucleation of any of these eyes should or could have been avoided. Once a secondary glaucoma complicates the conditions just demonstrated, the prognosis as to keeping a useful or comfortable eye is poor, and once a clinically well-founded suspicion of an intraocular growth has arisen, the eye is best removed."

W. S. REESE.

Uvea

SUBCHRONIC UVEOPAROTITIS (HEERFORDT). C. WESKAMP and E. ADROGUÉ, Arch. de oftal. de Buenos Aires **12**:319 (June) 1937.

After a few short and pertinent remarks on uveoparotitis, a case of Heerfordt's subchronic uveoparotitis (febris uveoparotidea subchronica) is reported. No hereditary or personal data with bearing on the disease in question were obtainable.

The patient was first seen by the authors in 1935. In 1929 he suffered from severe pain in the lumbar region, with pain in the testicles and swelling of the parotid glands. A diagnosis of mild parotitis, orchitis on the left side and pancreatitis was made. Symptomatic treatment was given. A year later the symptoms reappeared, lasting for seven years, with remissions. The symptoms consisted mostly of headaches (occipital), which were brought on by any undue effort or by psychic excitement, occurring sometimes several times a day and at others only every three or four days. Visual disturbances commenced in 1930, at first in the form of a central scotoma in the right eye, and gradually progressed to total loss of sight; later, visual disturbances appeared also in the left eye. Ophthalmoscopic examination showed the lesion in the right eye to consist of atrophy of the optic nerve with white subretinal masses; in places there was disappearance of the retinal pigment, with baring of sclerosed choroidal vessels. The diagnosis was proliferating, chorioretinitis subsequent to an original exudative choroiditis. In the left eye there were peripheral pigmentary changes, with narrowing of the visual field similar to that seen in retinitis pigmentosa.

Treatment with arsenicals, diathermy, tuberculin and other measures was entirely unsuccessful.

C. E. FINLAY.

Vitreous

TRAUMATIC SACKLIKE HERNIA OF THE VITREOUS INTO THE ANTERIOR CHAMBER AND CONSIDERATION OF THE LIMITING MEMBRANE OF THE VITREOUS. B. PAULA-SANTOS, Arch. d'opht. **53**: 876 (Dec.) 1936.

Prolapse of the vitreous into the anterior chamber takes place in two forms: the laminated, which is the most common, and the saccular. The author reports in detail a case of the latter type. Hess reported 6 similar cases in 1919 and was able to find only 2 other cases reported

in the literature up to that time. The author has found only 2 cases since 1920, making a total of 11. Haitz has suggested that in the first type the limiting membrane of the vitreous is ruptured, while in the second it is not. If Haitz's theory is correct it means that the hyaloid membrane is elastic. If this is so, the hernia of the vitreous should retract. The author suggests that the hyaloid membrane is ruptured in these cases at first and that a secondary membrane is formed about the hernia.

S. B. MARLOW.

DETACHMENT OF THE VITREOUS. V. ČAVKA, Arch. f. Ophth. **137:** 472 (Aug.) 1937.

Čavka describes one case of anterior and posterior detachment of the vitreous combined with other degenerative changes of the vitreous in an 8 year old patient with high myopia and a case of posterior detachment of the vitreous with formation of a hole in the hyaloid membrane following optic neuritis. For the study of the vitreous with the slit lamp he uses the contact glass of Koeppe and Koeppe's special corneal microscope, which is equipped with only one objective.

P. C. KRONFELD.

Therapeutics

ROENTGEN THERAPY IN TUBERCULOSIS OF THE ANTERIOR SEGMENT OF THE EYE. A. SUKONSTCHIKOVA and D. LUKIN, Vestnik oftal. **11:** 378, 1937.

The authors began using roentgen therapy (80 kilovolts in the secondary chain, 2 milliamperes and a 3 mm. aluminum filter) for diseases of the anterior segment of the eye in 1933. Up to date this treatment has been used in 2 cases of trachomatous pannus, 13 of tuberculous interstitial keratitis, 2 of keratoiritis, 13 of keratoscleritis, 17 of episcleritis and 3 of scleritis, making a total of 50 cases. All patients were affected by a severe recurrent process, for which no other treatment gave favorable results. The majority of the patients received from four to six treatments at weekly intervals. In a few instances the therapy had to be discontinued after the first treatment because of a severe reaction. An improvement was usually noticed after the second treatment.

The authors arrive at the following conclusions:

1. Roentgen therapy in the treatment of tuberculous diseases of the anterior segment of the eye is one of the most effective remedies.
2. The absorption of the tuberculous process occurs in a short time.
3. The treatment is most effective in cases of keratoscleritis.
4. The nodules in the sclera and episclera absorb completely without leaving scars.
5. Small doses, 10 per cent of a unit skin dose, or 60 roentgens, are preferable for insurance against late complications.

O. SITCHEVSKA.

Society Transactions

EDITED BY W. L. BENEDICT

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

JAMES W. WHITE, M.D., *Chairman*

Nov. 15, 1937

RUDOLF AEBLI, M.D., *Secretary*

MICROPHTHALMOS WITH THE FORMATION OF A CYST. DR. DONALD W. BOGART.

M. P. G., a boy 5 months of age, was admitted to the New York Eye and Ear Infirmary on Aug. 23, 1937, having been referred by Dr. Francis Shine. The child had been delivered normally, and after its birth it was noted that the right eye was apparently absent. In two weeks a "fleshlike" lump could be seen protruding through the palpebral fissure; it remained unchanged until the patient was seen. No other anomalies were noted. There was no history of consanguinity. A sister of the patient had a small eye with a coloboma of the iris and choroid and only light projection.

At the age of 5 months the child appeared to be normal and had an apparently normal left eye. The right eye was not present, being replaced by a mass of tissue within normal lids and foreshortened conjunctiva, from which protruded a bluish red cystic mass 1 cm. in diameter, which was semisoft and movable. The lids could be closed over the cystic mass. Roentgenograms of the skull and right orbit showed no abnormalities.

The baby was operated on under general anesthesia. The cyst and orbital contents were removed en bloc. Recovery was uneventful.

The sections disclosed a microphthalmic eye with a connected cyst, following the classic description. The walls of the cyst were composed of two coats: an outer fibrous one, continuous with the sclera, and an inner one, which consisted of more or less developed retina.

This condition shows distinct hereditary trends. Microphthalmic eyes may be divided into two classes: (1) those with no apparent congenital defect except the smallness of the globe and (2) those in which there is, in addition to smallness, an abnormality resulting from imperfect closure of the fetal fissure, as in this case.

DISCUSSION

DR. MORRIS ROSENBAUM: In 1931 I published a report of a case of bilateral anophthalmos. Dr. Doherty, who performed the operation at the Bellevue Hospital, never succeeded in finding any vestige of an eye. In 1936 I published the report of a case of unilateral microphthalmos with a cystic formation. In this case I enucleated the eye, and while I felt the cyst before the operation, when I took the eye out it was not

present; evidently it was so thin walled that it broke at the time of operation. In examining that eye I found degenerative changes, mostly bone formation, in the vitreous.

It is interesting to speculate as to the causation of anophthalmos and microphthalmos associated with a cystic formation. There are various theories concerning the formation of the cysts, and various experiments have been made by Ochi and Fred Hale. Hale published an article in 1935 wherein he reported anophthalmos in pigs from gilts maintained on a diet deficient in vitamin A. Ochi experimented on chicks with roentgen rays and other toxic substances and produced anophthalmos. In some of these cases I think the condition is due not only to vitamin A deficiency but to chromosome deficiency. When there is a chromosome deficiency somewhere in the gene, a maldevelopment occurs, either anophthalmos or microphthalmos, with cysts. Other causes may be hemorrhage during the fetal growth, amniotic strands or toxic substance.

COATS' DISEASE. DR. ROBERT M. OLIVER.

External deviation of the left eye and a normal right eye were found in an 8 year old boy. The vision in the left eye was 20/200. The disk was congested and indistinct. The nasal portion of the fundus was normal, and the remainder of the fundus presented white hyperplastic organizations, resembling popcorn, over which the retinal vessels followed the contour of each ridge and depression.

CONJUNCTIVITIS TULARENSIS. DR. DAVID H. WEBSTER.

This report will be published in full in a later issue of the ARCHIVES.

SARCOMA OF EYELID. DR. FRITZ BLOCH.

In a seaman 63 years old, who was otherwise healthy, two small tumors, about the size of small pears, developed twelve months previous to examination. The growths were covered with purplish ulcerated conjunctiva, giving the impression of granulating chalazions. The histologic examination showed mixed cell sarcoma originating in the tarsus.

CHORODIAL SCLEROSIS IN CORONARY ARTERIOSCLEROSIS. DR. MARTIN COHEN.

This paper was published in full in the April 1938 issue of the ARCHIVES, page 487.

JAMES W. WHITE, M.D., *Chairman*

Dec. 20, 1937

RUDOLF AEBLI, M.D., *Secretary*

RING SARCOMA: REPORT OF A CASE. DR. CLYDE E. MCDANNALD.

An Italian woman aged 24 was first seen on Feb. 9, 1935, having been referred from the office of Dr. Wilmot Allen. She gave a history of having had heterochromia since birth. The vision was reported to be

normal until six months prior to examination, at which time blurred vision and some inflammatory symptoms developed.

On examination the right eye was normal in every respect, and vision was 20/20 +. The vision in the left eye was 20/30; the cornea was clear, the anterior chamber was deep, and marked heterochromia of the iris was present. The pupil was mobile except from 7 to 9 o'clock, where it was definitely thickened. In this area, on the nasal side, considerable vascularization was present, and an apron of uveal pigment extended from the pigmented margin of the pupil over the iris. On transillumination the infiltrated area showed some thickening but no protrusion back of the iris. The media were clear, and the optic nerve showed marked glaucomatous cupping, with a small hemorrhage on the nasal side. The tension was 50 mm. of mercury (Schiötz). The Wassermann reaction was negative, and roentgenographic examination gave negative results.

The patient has been followed closely at monthly intervals up to the present time. The intraocular tension was only partially controlled by the use of pilocarpine hydrochloride, and in August 1935 the vision for the first time had notably diminished to 20/65. In the meantime the lesion had not changed except for increased vascularization and some enlargement of the apron of pigment.

The visual fields taken at intervals showed a progressive loss in the lower nasal quadrant. By February 1937 the vision had dropped to 20/200 +. There was no appreciable change in the appearance of the lesion. By August 1937 the vision had been further reduced to ability to count fingers at 10 inches (25 cm.), and the nerve head was completely cupped and showed a typical glaucomatous atrophy.

In November 1937, though the external appearance had not materially changed, transillumination disclosed a small papillary protrusion behind the iris extending from 7 to 8 o'clock, and the iridocorneal angle was completely blocked with pigmented tissue.

At the present time, the vision is limited to preception of hand movements, and the iris is depigmented and atrophic. The pupil is eccentric toward the nasal side, and uveal ectropion forms an apron extending from the pupillary margin to the angle, where the iris tissue is replaced by a vascularized, slightly grayish, fleshy mass.

DISCUSSION

DR. TRUMAN L. BOYES: In 1927 Dr. McDannald and I had the opportunity of examining a patient 62 years of age who previous to that time had normal vision in each eye. The pupil of the left eye was slightly pear shaped, and she stated that it had been so for two years. A year later Dr. McDannald saw her again. He presented her before the New York Ophthalmological Society, where it was agreed that the eye should be kept under observation. The following year, in 1929, the intraocular tension was increased. There was a growth in the iris and ectropion of the uvea from 4 to 6 o'clock, where the iris had become thickened. The tension was elevated to 60 mm. of mercury when Dr. McDannald presented the patient a second time before the society. The consensus then was that the eye should be removed. That was four years after the first examination. The sections showed a sarcoma of the iris and ciliary body.

DR. MARK SCHOENBERG: I should like to ask whether roentgenograms have revealed metastases in some other part of this patient's body and if the urine contains melanin.

DR. CLYDE E. MCDANNALD: The patient has not had a general roentgenographic examination. I plan to have that done, and I am checking up on the Wassermann reaction. When the patient was first seen she had a negative Wassermann reaction.

NOTE.—The left eye was enucleated on March 2, 1938. The diagnosis was confirmed.

ETIOLOGY AND TREATMENT OF KERATOCONUS. DR. ARTHUR ALEXANDER KNAPP.

This report on the causation, pathologic process and treatment of keratoconus is based on two sets of observations: (1) animal experimentation, in which pathologic examinations of the affected eyes were made, and (2) the improvement observed in man after the indicated therapy had been given.

In a previous paper, from the College of Physicians and Surgeons, Columbia University, the department of pharmacology, Blackberg and I reported the consistent production of keratoconus in dogs fed a diet deficient in vitamin D and low in calcium. In a more recent paper, we published a report on the pathologic process in the fibrous tunic of the affected eyes. (These pathologic changes were reviewed with the aid of slides.)

Twelve patients with keratoconus are included in the present study. Eight are present for examination by members of the section, and 1 is shown so that the condition of his right cornea may be noted before the institution of therapy. The remaining 11 patients have been treated for periods ranging from three months to three years. Various degrees of involvement of the cornea are present. Plaster casts of several conical corneas before and after treatment are on display.

The treatment consisted of vitamin D, in the form of viosterol (10,000 units of vitamin D per gram), and calcium, in the form of mineral mixture tablets composed of alfalfa ash, dicalcium phosphate and edible bone meal.

The subjective and objective improvement have been studied. (Slides were shown depicting this phase of the study.) To gage the objective improvement, plaster casts, retinoscopic examination under cycloplegia and microscopic and macroscopic examinations of the cornea have been utilized. Measurements of the molds were made by Professor De Zafra, of the engineering department of New York University. Without exception, all of the patients who were treated improved; the cones were flattened.

A further study is being made.

DISCUSSION

PROF. CARLOS DE ZAFRA: In order to measure the plaster cast molds, a "toolmakers' microscope" was used. At the College of Engineering of New York University is located the precision measurement and gage checking laboratory of the United States War Department, where a

number of instruments for various precision measurements are available. When Dr. Knapp first came to me, the problem was to mount these plaster casts so as to get the desired measurements. First we tried projecting them against a screen with a screw thread comparator, but owing to the narrowness of the light beam the shadow on the screen did not represent the full width of the cast; so we resorted to a toolmakers' microscope, the table of which is represented in the accompanying illustration. To this table (*A*), which is mounted on ball bearings, are attached two micrometers, *M* and M_1, graduated in ten-thousandths of an inch, for measuring lateral and longitudinal movement of the table.

Back of the center of the table was a Hoke precision gage block, *B*, of dimensions given by Dr. Knapp according to the diameter of the specimen to be examined. A gage makers' precision square, *CC*, was used to line up the gage block with the axes of the table. Touching this gage block was a plaster cast, *D*, making contact as at *O*. When one

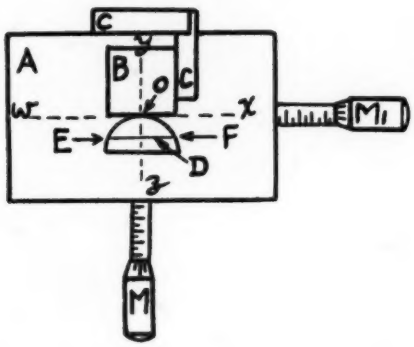

Diagram of the table of a toolmakers' microscope

looked through the microscope, the cross hairs, *wx* and *yz*, were brought in coincidence at *O*, the point of tangency of the plaster cast and the gage block. A reading on micrometer *M* was then recorded, which became the basic, or reference, measurement. Then the intersection of the cross hairs was made coincident with the point *E* of the plaster cast. The difference between the measurement here recorded and the basic measurement represented the height of the apex. However, because of the difficulty of placing the plaster cast with certainty that the circle diameter *EF* was absolutely parallel with the reference surface of the gage block *B*, the cross hairs of the microscope were similarly brought in coincidence with the point *F* of the circle, and the measurement thus indicated on micrometer *M* was deducted from the basic measurement and the height of the apex gain determined. By virtue of the fineness of the measurements (to the ten-thousandth part of an inch), any inaccuracy in setting the circle line or diameter *EF* parallel to the face of the gage block was corrected by taking the mean of the two readings. This figure, in decimals of an inch, is readily convertible into millimeters by multiplying by the conversion factor 25.4.

CONTRAST MEDIUMS IN ORBITAL ROENTGENOGRAPHY. DR. MURRY A. LAST.

This preliminary report is based on an investigation of the use of air injected into the orbits for roentgenographic and fluoroscopic studies of normal and pathologic structures, including foreign bodies. Stereoscopic pneumograms showed the outlines of the displaced globes, the accessory structures and dense shadows which corresponded to neoplasms, the presence of which was later confirmed by operation. These growths included an epithelial cyst, a myxofibrosarcoma and a thyrogenic metastatic tumor of the orbit. An inflammatory pseudotumor in which the orbital vessels showed an almost complete endarteritic obliteration was revealed in the roentgenograms of the orbit after the injection of air as a diffuse density about the globe. In all these cases roentgenograms made by the usual methods were reported to be normal.

The technic is simple. By means of a sterile syringe, air is introduced through the lids into the deep orbital fat, the normal structures and areas thought to be pathologic being avoided. The use of repeated aspirations to avoid embolic phenomena, the slow introduction of the air to avoid pain and injection of a sufficient amount of air to obtain a marked emphysematous proptosis are important. No untoward symptoms were present.

Carbon dioxide or oxygen may be used. Colloidal thorium dioxide should not be used because of its poor diffusibility, its retention and its radiant powers. Iodized poppyseed oil 40 per cent causes fibrosis and was not employed in this study.

Microscopic sections of the fibrosarcoma showed ocular changes resulting from localized pressure, which had caused inward bulging and corrugation folds of the retina.

THE LYMPHATICS OF THE ORBIT. DR. O. V. BATSON.

The lymphatic drainage of the lids, the conjunctiva and the lacrimal gland has been carefully studied, and the various investigators are in substantial agreement as to the anatomic structure. In a general way, the regional lymph glands for these structures are either in the area about the parotid gland or in the area about the submaxillary salivary gland. The rich lymph capillary plexuses of the lids and conjunctiva near the inner canthus, the medial fourth of the upper lid and the medial three fourths of the lower lid drain into collectors, which drain to the submaxillary region. The remaining portions of the lids and of the conjunctiva, together with the lacrimal gland, contain networks which drain to the region of the parotid gland.

It has been shown by a number of workers that material experimentally deposited in the subarachnoid space of the brain will travel out of the cranium along the optic nerve sheath as far as the globe. This extent and limitation of travel are in accord with the microscopic anatomic structure of the nerve sheath and the subarachnoid space.

There has been much doubt expressed as to the presence of true lymphatics within the globe or in the retroglobar tissues. The major evidence is against their presence. Nuel and Benoit in 1899 were able to demonstrate ink granules in the iris after the injection of ink into the anterior chamber of the eye. Definite channels were not demonstrated, however.

It was hoped that some of the roentgen opaque materials might offer a method of investigating this problem. Colloidal thorium dioxide when injected intradermally may be followed to the regional lymph nodes in roentgenograms taken a few moments later. Iodized oils injected into the pleural cavity can be seen in the mediastinal lymphatic and nodes within a few hours.

Repeated injections were made behind the globe in a series of dogs. Iodized oil and colloidal thorium dioxide were used. After an initial period of edema and capillary injection, the tissues regained their normal appearance. The roentgenograms showed that the injected material distributed itself along the muscles and became rather generally disseminated in the orbit. At no time could lymphatic shadows be made out in the roentgenogram. Roentgenographic examination of the spleen and liver in the intact animal and in the excised specimens showed no increased densities.

In animals observed for a period of several weeks, densities were seen to appear in lymph glands lying in the neck along the carotid artery. No connecting channels could be observed. In six weeks the shadow was definite.

Injections into the anterior chamber had to be terminated because of the intense inflammatory response. No early migration of the roentgen opaque substance was seen, nor was it to be expected on a basis of the structure of the parts.

These experimental injections tend to confirm the view that true lymphatics do not exist in the orbital tissues. The slow appearance of the injected material in the lymph nodes of the neck is no doubt due to the migration to the regional lymphatic glands of wandering cells loaded with thorium dioxide.

Book Reviews

The Science of Seeing. By Matthew Luckiesh, D.Sc., D.E., and Frank E. Moss, E.E. Price, $6. Pp. 548, including index. New York: D. Van Nostrand Co., Inc., 1937.

This work is a detailed exposition of a relatively new science, which the authors choose to call the "science of seeing." They make a distinction at the outset between the "science of vision" as practiced by the ophthalmologist, limiting it largely to the correction of optical and muscular anomalies, and the "science of seeing," which takes in all the complex elements involved in the use of visual sense. These include primarily the factors of lighting, but also other elements, such as speed, accuracy and ease of seeing, the causes of general and special fatigue and work efficiency.

The whole subject is treated more from the standpoint of the illuminating engineer, and emphasis is placed on the study of man as a "seeing machine." This term and concept predominates throughout. The authors elaborate on the premise that prolonged near work and reduced inadequate (indoor) illumination are the twin curses of modern civilization. They then proceed at great length to prove their premise by biologic considerations, philosophic deductions and the results of extensive experimental work. They produced a number of ingenious devices for experimental work on the act of seeing. Their experiments embraced studies on the various aspects of light and lighting, effects of contrasts, pupillary size and reactions, changes in accommodative and convergence amplitudes, the rate of blinking and even changes in the heart rate. The results are presented in numerous tables, graphs, equations and statistical reports.

They make out a fairly good case for their contention that the general level of illumination prevalent at present is much too low for the optimum efficiency and comfort of the "human seeing machine." Adopting the term "humanitarian foot candles" for illumination, which in addition to providing adequate light for efficient seeing is also conducive to visual and bodily ease, they claim "that at least 100 ft. candles are desirable for reading under usual conditions," and for this they advocate a system of general lighting plus supplementary lighting, the general lighting to be not less than one tenth in candle power of the supplementary lighting. They arrive at these results largely from what they consider objective findings, since, as they repeatedly say, "Human beings are very poor judges of seeing conditions. They are poor seeing meters. . . ."

The authors show little respect for the ordinary findings and facts of physiologic optics, largely because these facts were obtained for the most part by subjective methods. Statements like "The common error of building an argument upon the narrow viewpoint and the incompleteness of knowledge of physiologic optics should not be made" are rather frequent.

But in their own more scientific approach they repeatedly come up against these same elements of subjective uncertainty. One can readily agree with the authors that it is best to have objective "scientific" methods of approach in the study of vision and seeing. But ultimately seeing is a complex physiopsychologic activity; as the authors themselves say, "The eyes—the visual sense—the entire human seeing machine are necessarily a part of any demonstration of seeing. The inconstancy of these factors makes it necessary to obtain many observations . . . to yield dependable results." And it may be added that these dependable results will still not be reducible to objective machine measurements. Here and there in the book there crops out a sort of undercurrent clash between the desire and the appearance of straight objective findings and the somewhat reluctant admission of the inevitable subjective elements involved.

The authors describe several instruments of their own invention, including the sensitometer. This measures subjectively "sensitivity difference," or the appreciation of "brightness difference." They claim that an appraisal of visual efficiency as well as the correction of refractive errors by the "brightness difference" method of the sensitometer is superior to the methods ordinarily used by the Snellen visual acuity charts. Claims are made with reference to the better control of accommodation monocularly and binocularly. With another device, the L. M. visibility control, it is said that greater refinement in the study of the elements of binocular vision and orthoptics is possible. While much of this sounds plausible, the whole treatment of the purely refractive, accommodative and binocular phases of vision does not have the sure scientific touch which characterizes the treatment of the other topics. One wonders where the authors got the information which leads them to say, "Although the subjective and objective techniques are often considered to be competitive methods for revealing and measuring refractive errors it would seem more appropriate to regard one as supplementary to the other." It is certainly the standard practice among ophthalmologists to regard the subjective and objective methods as supplementary and not as "competitive." More reliance is sometimes placed on one method than on the other, but not in a "competitive" sense.

In style, the work is somewhat verbose and repetitious. It could be considerably condensed and still retain all the essential facts and findings. Some of the diagrams and graphs are difficult to understand and will be especially irksome to physicians not thoroughly conversant with such methods.

But with all that there is a good deal of sound, informative material in the book for the practicing ophthalmologist. He will be impressed with the importance of the elements of brightness and brightness difference for good and comfortable seeing, as against mere light intensity. And with reference to light intensity, he will be impressed with the finding, though he may be vaguely aware of it, that in an actual experiment with a group of persons wearing glasses the corrected vision under low illumination was decidedly worse than the uncorrected vision under high illumination.

As the book is written by lighting engineers, overenthusiasm in the direction of overlighting may be expected. But the ophthalmologist

who wishes to render his patients a full ocular service will find in the book much useful information of a general nature and many helpful suggestions which will aid him in advising his patients relative to their seeing habits.

JOSEPH I. PASCAL.

Twenty-Third Annual Report of the Ophthalmic Hospitals Section for 1935. Ministry of Public Health, Egypt. Price, 12 piasters. Cairo: Government Press, 1937.

The annual report for 1935 shows that 5 additional ophthalmic branches were opened in the general hospitals in Egypt, the total number of ophthalmic units being 69. During the year there were 1,034,986 new patients and 32,623 inpatients, and 334,866 operations were performed. The percentage of patients who were found blind in one or both eyes was 6. Acute ophthalmia was responsible for 80 per cent of the blindness, and the gonococcus was still the predominating factor in the production of acute ophthalmia. Of 10,412 pupils examined in the school clinics, 99 per cent were found to be suffering from trachoma in its various stages; in 43 per cent the condition was serious.

A list of interesting conditions seen in the various units of the section is included. A report of the meeting of the Ophthalmological Society of Egypt is also given.

Of 128,138 operations performed, 1,920 were for senile cataract, 345 for soft cataract and 99,474 for trichiasis or entropion. The other operations, 26,400 in number, consisted of 6,402 excisions of the lacrimal sac; 1,295 trephinings of the cornea and sclera, with iridectomy; 12 operations for detachment of the retina, and 24 tenotomies, with advancement. Pathologic reports show that 1,081 specimens were examined.

The causes of blindness in 65,971 cases were analyzed as follows: complications following conjunctivitis, 50,000 cases; atrophy of the optic nerve, 160, detachment of the retina, 126; retinitis pigmentosa, 43, monocular glaucoma, 3,440, and binocular glaucoma, 2,555.

ARNOLD KNAPP.

Elementi di biomicroscopia oculare. By G. Sala. Price, 50 liras. Pp. 206, with 88 illustrations. Bologna: Licinio Cappelli.

This is the first manual of biomicroscopy of the eye for the use of the practicing oculist that has appeared in Italian. The book is divided in two parts. In the first part the Zeiss slit lamp is described minutely, and the principles of the biomicroscopic examination are explained. The technic of this examination, with the different illuminations used, is well described, and useful advice is given for the interpretation of the images seen.

The second part deals with the description of the different parts of the eye, and the chapters are divided according to their anatomic precedence. Gonioscopy is mentioned briefly.

The book is plainly written and can easily be understood by the beginner. It is rich in illustrations, several of which were made by

the author; they help to give in a schematic form a clear picture of the progress made with this objective examination of the eye.

The bibliography is extensive, with more than 400 references to Italian and foreign authors. This book will be of great aid in making the use of the slit lamp popular, and it deserves to be on the bookshelf of every oculist.

V. La Rocca.

Neurology. By Roy R. Grinker, M.D. Second edition. Price, $8.50. Pp. 999. Springfield, Ill.: Charles C. Thomas, Publisher, 1937.

The first edition of this textbook was reviewed in the Archives in November 1934 (vol. 12, p. 796). The second edition consists of a revision of the first, with the addition of new matter on the vegetative nervous system, on localization in the cerebral cortex and on electroencephalography. The chapter on infections has been rearranged, and new treatment for migraine, myasthenia gravis and myotonia congenita, material on vitamin deficiency in neuritis and new data on the relation of the cerebellum to the cortex have been added. These additions bring the book up to date and, together with the many admirable features which characterized the previous presentation of the subject-matter in the first edition, make it an unusually good textbook which should appeal to a steadily increasing group of readers.

Arnold Knapp.

Anuario Médico Social de Cuba. Price, $5. Pp. 684. Havana, Cuba: Ucar, García and Cía, 1937.

Revista cubana de oto-neuro-oftalmiatria, directed in Havana by Dr. Thomás R. Yanes, has made possible the edition of this work, which contains not only interesting data on the medical development of Cuba but the addresses of all the practicing physicians in this republic, with their names, their specialties, the year of their graduation, the universities from which they have graduated and other data.

The work is completely original. It is not a simple guide or medical directory; personal data are also given for each physician, which include several admirable and amusing caricatures.

This year book more than fulfils its purpose and will be found useful in many ways.

Arnold Knapp.

External Diseases of the Eye. By D. T. Atkinson, M.D. Pp. 718, with 494 engravings. Second edition. Price, $8. Philadelphia: Lea & Febiger, 1937.

The first edition of this book was reviewed by Dr. Sanford R. Gifford in the September 1934 issue of the Archives, page 461.

The second edition differs slightly from the first. The author has included a paragraph on conjunctival myiasis and one on iridencleisis for glaucoma. A page has been devoted to the discussion, in general terms, of orthoptic training. About one and a half pages cover the slit lamp appearance of the crystalline lens.

The illustrations still constitute the best feature of the book.

O. P. Perkins.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

Honorary President: Prof. F. de Lapersonne, 217 Fauborg St. Honoré, Paris.
President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the President, Dr. P. Bailliart.

International Ophthalmologic Congress

Secretary: Dr. E. Marx, Costzeedijk 316, Rotterdam, Netherlands.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 33 Welbeck St., London, W., England.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Dr. Stewart Duke-Elder, 59 Harley St., London, W. 1.
Secretary: Dr. Thomasina Belt, 13 Mitchell Ave., Jesmond, Newcastle-on-Tyne.
Place: Plymouth. Time: July 20-22, 1938.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

German Ophthalmological Society

President: Prof. W. Lohlein, Berlin.
Secretary: Prof. E. Engelking, Heidelberg.
Place: Heidelberg. Time: July 4-6, 1938.

Midland Ophthalmological Society

President: Dr. T. E. Ashdown Carr, 34 Charnwood St., Derby, England.
Secretary: T. Harrison Butler, 81 Edmund St., Birmingham, England.
Place: Birmingham and Midland Eye Hospital.

Ophthalmological Society of Egypt

President: Prof. Dr. Mohammed Mahfouz Bey, Government Hospital, Alexandria.
Secretary: Dr. Mohammed Khalil, 4 Baehler St., Cairo.
All correspondence should be addressed to the Secretary, Dr. Mohammed Khalil.

Ophthalmological Society of the United Kingdom

President: Mr. Gordon M. Holmes, 9 Wimpole St., London, W. 1.
Secretary: Mr. H. B. Stallard, 35 Harley St., London, W. 1.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.
Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.
Time: July 7-9, 1938.

PALESTINE OPHTHALMOLOGICAL SOCIETY

President: Dr. Arieh Feigenbaum, Abyssinian Str. 15, Jerusalem.
Secretary: Dr. E. Sinai, Tel-Aviv.

POLISH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warsaw.
Place: Lindley'a 4, Warsaw.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Dr. Ransom Pickard, 31 E. Southernhay, Exeter, England.
Secretary: Dr. A. Rugg-Gunn, 35 Harley St., London, W. 1.

SOCIÉTÉ FRANÇAISE D'OPHTALMOLOGIE

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris, 7è.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. K. G. Ploman, Stockholm, Sweden.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

TEL-AVIV OPHTHALMOLOGICAL SOCIETY

President: Dr. D. Arieh-Friedman, 96 Allenby Str., Tel-Aviv.
Secretary: Dr. Sadger Max, 9 Bialik Str., Tel-Aviv.

TSINAN OPHTHALMOLOGICAL SOCIETY

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung.
Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. Derrick T. Vail Jr., 441 Vine St., Cincinnati.
Place: San Francisco. Time: June 13-17, 1938.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Washington, D. C. Time: Oct. 9-14, 1938.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. Frederick H. Verhoeff, 243 Charles St., Boston.
Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.
Place: San Francisco. Time: June 9-11, 1938.

CANADIAN OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Gordon M. Byers, 1458 Mountain St., Montreal.
Secretary-Treasurer: Dr. Alexander E. MacDonald, 421 Medical Arts Bldg., Toronto.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Secretary: Miss Regina E. Schneider, 50 W. 50th St., New York.

SECTIONAL

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. L. C. Gardner, 11 N. Main St., Fond du Lac.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.
Place: Marshfield. Time: May 1938.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. Edwin B. Goodall, 101 Bay State Rd., Boston.
Secretary-Treasurer: Dr. William P. Beetham, 5 Bay State Rd., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. William D. Donoher, 1930 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.
Place: Victoria, B. C. Time: June 21-24, 1938.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. A. W. Howe, 740 St. Helens Ave., Tacoma, Wash.
Secretary-Treasurer: Dr. Purman Dorman, 1115 Terry Ave., Seattle.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month, except June, July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Frank W. Broderick, 501 Central Trust Bldg., Sterling, Ill.
Secretary-Treasurer: Dr. Thorsten E. Blomberg, 501–7th St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Secretary-Treasurer: Dr. Andre Cortopassi, 703 Second National Bank Bldg., Saginaw, Mich.
Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. J. H. Judd, 107 S. 17th St., Omaha, Neb.
Secretary-Treasurer: Dr. J. C. Decker, 515 Frances Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Grady E. Clay, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. John Hunter McRae, 26 Sheldon Ave., S. E., Grand Rapids.
Secretary-Treasurer: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. M. Harris, Johnstown.
Secretary-Treasurer: Dr. C. Wearne Beals, Weber Bldg., DuBois.
Place: Johnstown. Time: May 19, 1938.

STATE

Colorado Ophthalmological Society

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. John C. Long, 324 Metropolitan Bldg., Denver.
Place: Capitol Life Bldg., Denver. Time 7:30 p. m., third Saturday of the month, October to April, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. Charles T. Flynn, 41 Trumbull St., New Haven.
Secretary-Treasurer: Dr. Shirley H. Baron, 309 State St., New London.
Place: New London. Time: February 1938.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. John King, Thomasville.
Secretary-Treasurer: Dr. Mason Baird, 384 Peachtree St., Atlanta.
Place: Augusta. Time: May 1938.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. C. W. Rutherford, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Marlow W. Manion, 23 E. Ohio St., Indianapolis.
Place: Indianapolis. Time: First Wednesday in April.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. H. Lamb, American Bank Bldg., Davenport.
Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.
Place: Davenport.

Louisiana-Mississippi Ophthalmological and Otolaryngological Society

President: Dr. C. A. McWilliams, G. and S. I. R. R. Bldg., Gulfport, Miss.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Place: New Orleans. Time: Spring, 1938.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. F. N. Smith, Grand Rapids Clinic, Grand Rapids.
Secretary: Dr. Dewey R. Heetderks, 26 Sheldon Ave., S. E., Grand Rapids.

Minnesota Academy of Ophthalmology and Otolaryngology

President: Dr. Walter E. Camp, 1918 Medical Arts Bldg., Minneapolis.
Secretary-Treasurer: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Roy Grigg, Bozeman.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. Oram Kline, 414 Cooper St., Camden.
Secretary: Dr. James S. Shipman, 542 Cooper St., Camden.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Marvin F. Jones, 121 E. 60th St., New York.
Secretary: Dr. Algernon B. Reese, 73 E. 71st St., New York.
Time: May 10, 1938.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. M. Lilly, 302 Old St., Fayetteville.
Secretary-Treasurer: Dr. Frank C. Smith, 106 W. 7th St., Charlotte.
Place: Charlotte. Time: October.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. Rosenberger, 221–5th St., Bismarck.
Secretary-Treasurer: Dr. F. L. Wicks, 514–6th St., Valley City.
Place: Bismarck. Time: May 1938.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Society Library, Providence. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. George Truluck, Orangeburg.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Kate Savage Zerfoss, 165–8th Ave., N., Nashville.
Secretary-Treasurer: Dr. W. D. Stinson, 805 Medical Arts Bldg., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. T. E. Fuller, 100 W. Board St., Texarkana, Texas.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. M. H. Hood, 505 Washington St., Portsmouth.
Secretary-Treasurer: Dr. Charles T. St. Clair, 418 Bland St., Bluefield, W. Va.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. James A. Fisher, 501 Grand Ave., Asbury Park.
Secretary: Dr. William F. McKim, 488 Sanford Ave., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

Akron Academy of Ophthalmology and Otolaryngology

President: Dr. L. E. Brown, Second National Bldg., Akron.
Secretary-Treasurer: Dr. C. R. Anderson, 106 S. Main St., Akron.
Time: First Monday in January, March, May and November.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. J. Mason Baird, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. Alton V. Hallum, 478 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. Time: Second Friday of each month from October to May.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Henry F. Graff, 513 N. Charles St., Baltimore.
Secretary: Dr. Frank B. Walsh, Wilmer Institute, Johns Hopkins Hospital, Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. Walter V. Moore, 1 Nevins St., Brooklyn.
Secretary-Treasurer: Dr. Mortimer A. Lasky, 1 Nevins St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Ivan J. Koenig, 40 North St., Buffalo.
Secretary-Treasurer: Dr. Meyer H. Riwchun, 367 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Georgiana Dvorak-Theobald, 715 Lake St., Oak Park.
Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Webb Chamberlin, 7405 Detroit Ave., Cleveland.
Secretary: Dr. Walter H. Rieger, Hanna Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. Paul Moore, Republic Bldg., Cleveland.
Secretary: Dr. G. Leslie Miller, 14805 Detroit Ave., Cleveland.
Time: Second Tuesday in October, December, February and April.

COLLEGE OF PHYSICIANS, PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

COLUMBUS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

Chairman: Dr. C. E. Silbernagle, 247 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. D. M. Johnson, 327 E. State St., Columbus, Ohio.
Place: Deshler Wallick Hotel. Time: 6 p. m., first Monday of each month.

CORPUS CHRISTI EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. Edgar G. Mathis, 416 Chaparral St., Corpus Christi, Texas.
Secretary: Dr. E. King Gill, 416 Chaparral St., Corpus Christi, Texas.
Time: Second Thursday of each month from October to May.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Lester H. Quinn, 4105 Live Oak, Dallas, Texas.
Secretary: Dr. J. Dudley Singleton, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

DETROIT OPHTHALMOLOGICAL CLUB

Chairman: Members rotate alphabetically.
Secretary: Dr. William Fowler, 1424 Maccabee Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

President: Dr. J. D. Carroll, 102–3d St., Troy, N. Y.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth, Texas.
Secretary-Treasurer: Dr. Charles R. Lees, 306 W. Broadway, Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

GRAND RAPIDS EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 116 E. Fulton St., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

HOUSTON ACADEMY OF MEDICINE, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. L. Allen, 1215 Walker Ave., Houston, Texas.
Secretary: Dr. George C. Farrish, 1625 Main St., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. C. P. Clark, 24 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. O. S. Gilliland, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. John S. Knight, 1103 Grand Ave., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. F. C. Hertzog, 117 E. 8th St., Long Beach, Calif.
Secretary-Treasurer: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Clifford B. Walker, 427 W. 5th St., Los Angeles.
Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

LOUISVILLE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. S. Bumgardner, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. Max Bornstein, Heyburn Bldg., Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Earle Breeding, 1801 I St., N. W., Washington.
Secretary: Dr. Elmer Shepherd, 1606–20th St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sonders, Medical Arts Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. John E. Mulsow, 231 W. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. John B. Hitz, 411 E. Mason St., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

MINNEAPOLIS OPHTHALMOLOGICAL SOCIETY

Chairman: Each member, in alphabetical order.
Secretary: Dr. M. C. Pfunder, 645 Medical Arts Bldg., Minneapolis.
Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

MONTGOMERY COUNTY MEDICAL SOCIETY

Chairman: Dr. P. H. Kilbourne, Fidelity Bldg., Dayton, Ohio.
Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.
Place: Van Cleve Hotel. Time: 6:30 p. m., monthly, first Tuesday from October to June, inclusive.

MONTREAL OPHTHALMOLOGICAL SOCIETY

President: Dr. Stuart Ramsay, 1496 Mountain St., Montreal, Canada.
Secretary: Dr. J. Rosenbaum, 1396 St. Catherine St., W., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Guy Maness, 119–7th Ave., Nashville, Tenn.
Secretary-Treasurer: Dr. Andrew Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Secretary-Treasurer: Dr. H. F. Brewster, 837 Gravier St., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. James W. White, 15 Park Ave., New York.
Secretary: Dr. Rudolf Aebli, 30 E. 40th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

President: Dr. Mark J. Schoenberg, 1160 Park Ave., New York.
Secretary: Dr. Jesse Stark, 45 Park Ave., New York.
Place: Squibb Hall, 745–5th Ave. Time: 7 p. m., first Monday of each month from October to May, inclusive.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. A. J. Young, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. Delbert K. Judd, 1020 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

OPHTHALMOLOGICAL SOCIETY OF THE UNIVERSITY OF PITTSBURGH

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Time: Second Monday in November, January, March and May.

PASSAIC-BERGEN OPHTHALMOLOGICAL CLUB

President: Dr. John S. Van Winkle, 297 Broadway, Paterson, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 340 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Glendon E. Curry, Westinghouse Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

RICHMOND OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. N. H. Turner, 200 E. Franklin St., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. R. E. Elliott, 78 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Raphael Farber, 280 Monroe Ave., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. Lawrence T. Post, 508 N. Grand Blvd., St. Louis.
Secretary: Dr. Leslie Charles Drews, 508 N. Grand Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Russell Fletcher, 490 Post St., San Francisco.
Secretary: Dr. Avery Morley Hicks, 490 Post St., San Francisco.
Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except May, June, July and December.

SHREVEPORT EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. A. Wilkinson, Medical Arts Bldg., Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Raymond A. Lower, 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. Louis A. Parsell, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

SYRACUSE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Gordon D. Hoople, 110 Medical Arts Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 212 Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: A. Lloyd Morgan, Medical Arts Bldg., Toronto, Canada.
Secretary: Dr. W. R. F. Luke, Medical Arts Bldg., Toronto, Canada.
Place: Academy of Medicine, 13 Queen's Pk. Time: First Monday of each month, November to April.

WASHINGTON, D. C., OPHTHALMOLOGICAL SOCIETY

President: Dr. G. Victor Simpson, 1710 Rhode Island Ave., N. W., Washington, D. C.
Secretary-Treasurer: Dr. Frank D. Costenbader, 1726 I St., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday in November, January, March and May.

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